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Logging in to Dialog

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DIALOG INFORMATION SERVICES
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ENTER PASSWORD:
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Dialog level 05.22.00D

Last logoff: 17jun08 15:49:48
Logon file405 19jun08 10:41:13
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                    ***
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***File 50, CAB Abstracts
***File 162, Global Health
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SYSTEM:HOME

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Cost is in DialUnits
Menu System II: D2 version 1.8.0 term=ASCII
                     *** DIALOG HOMEBASE(SM) Main Menu ***

 Information:
  1.  Announcements (new files, reloads, etc.)
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  3.  Help in Choosing Databases for Your Topic
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 service.  Enter a BEGIN command plus a file number to search a
database
(e.g., B1 for ERIC).
? b 410

       19jun08 10:41:13 User226352 Session D1072.1
            $0.00    0.265 DialUnits FileHomeBase
     $0.00  Estimated cost FileHomeBase
     $0.00  Estimated cost this search
     $0.00  Estimated total session cost   0.265 DialUnits

File 410:Dialog Comm.-of-Interest Newsletters 2008 /Mar
       (c) 2008 Dialog

      Set  Items  Description
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HILIGHT set on as ''
HILIGHT set on as ''
? b biochem
>>>          76 is unauthorized
>>>1 of the specified files is not available
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            $0.00    0.115 DialUnits File410
     $0.00  Estimated cost File410
     $0.03  TELNET
     $0.03  Estimated cost this search
     $0.03  Estimated total session cost   0.380 DialUnits

SYSTEM:OS  - DIALOG OneSearch
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  File   5:Biosis Previews(R) 1926-2008/Jun W3
         (c) 2008 The Thomson Corporation
  File   6:NTIS 1964-2008/Jun W4
         (c) 2008 NTIS, Intl Cpyrght All Rights Res
  File  24:CSA Life Sciences Abstracts 1966-2008/Mar
         (c) 2008 CSA.
  File  34:SciSearch(R) Cited Ref Sci 1990-2008/Jun W4
         (c) 2008 The Thomson Corp
  File  40:Enviroline(R) 1975-2008/Apr
         (c) 2008 Congressional Information Service
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         (c) 2008 CSA.
  File  45:EMCare 2008/Jun W3
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         (c) 2008 CAB International
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have changed. See HELP NEWS50 for information.
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         (c) 2008 BLDSC all rts. reserv.
  File  71:ELSEVIER BIOBASE 1994-2008/Jun W2
         (c) 2008 Elsevier B.V.
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*File  73: The file has been reprocessed.
  File  98:General Sci Abs 1984-2008/May
         (c) 2008 The HW Wilson Co.
  File 103:Energy SciTec 1974-2008/Apr B2
         (c) 2008 Contains copyrighted material
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         (c) 2007 CSA.
*File 136: This file is closed.
  File 143:Biol. & Agric. Index 1983-2008/Apr
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  File 144:Pascal 1973-2008/Jun W2
         (c) 2008 INIST/CNRS
  File 155:MEDLINE(R) 1950-2008/Jun 18
         (c) format only 2008 Dialog
*File 155: MEDLINE has reloaded. Please see HELP NEWS 155
for details.
  File 156:ToxFile 1965-2008/Jun W2
         (c) format only 2008 Dialog
*File 156: ToxFile has been reloaded. Accession numbers have
changed.
  File 162:Global Health 1973-2008/May W4
         (c) 2008 CAB International
*File 162: The file has been reloaded and accession numbers have
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  File 172:EMBASE Alert 2008/Jun 13
         (c) 2008 Elsevier B.V.
  File 305:Analytical Abstracts 1980-2008/Apr W4
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         (c) 2008 Royal Soc Chemistry
*File 305: Alert feature enhanced for multiple files, duplicate
removal, customized scheduling.  See HELP ALERT.
  File 369:New Scientist 1994-2008/May W2
         (c) 2008 Reed Business Information Ltd.
  File 370:Science 1996-1999/Jul W3
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*File 370: This file is closed (no updates). Use File 47 for more
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  File 393:Beilstein Database - Abstracts 2007/Q4
         (c) 2008 Beilstein GmbH
  File 399:CA SEARCH(R) 1967-2008/UD=14825
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      Set  Items  Description
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leuX or pathogenicity(w)island)
          999613  ATTENUAT?
           30122  AVIRULENT
         1153992  VACCIN?
          441715  SALMONELLA
           51654  PAI
             234  LEUX
          302725  PATHOGENICITY
          466932  ISLAND
           10789  PATHOGENICITY(W)ISLAND
      S1     581  (ATTENUAT? OR AVIRULENT OR VACCIN?) AND SALMONELLA
AND
                  (PAI OR LEUX OR PATHOGENICITY(W)ISLAND)
? rd s1

>>>Duplicate detection is not supported for File 393.

>>>Records from unsupported files will be retained in the RD set.
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         8209634  PY>2006
      S3     139  S2 NOT PY>2006
? t s3/7/1-10
>>>Format 7 is not valid in file 143

 3/7/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
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```

19388451 BIOSIS NO.: 200700048192
Acute systemic inflammation upregulates endogeneous fibrinolysis: a counterbalancing mechanism?
AUTHOR: Dima I (Reprint); Vlachopoulos C; Aznaouridis K; Ioakeimidis N;
Vasiliadou C; Alexopoulos N; Tousoulis D; Stefanadis C
AUTHOR ADDRESS: Hippokrateion Hosp, Athens Med Sch, Dept Cardiol 1, Athens,
Greece**Greece
JOURNAL: European Heart Journal 27 (Suppl. 1): p77 AUG 2006 2006
CONFERENCE/MEETING: World Congress of Cardiology Barcelona, SPAIN September 02 -06, 2006; 20060902
ISSN: 0195-668X
DOCUMENT TYPE: Meeting; Meeting Poster
RECORD TYPE: Citation
LANGUAGE: English

3/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

19287886 BIOSIS NO.: 200600633281
Virulent Salmonella enterica serovar typhimurium evades adaptive immunity by preventing dendritic cells from activating T cells
AUTHOR: Tobar Jaime A; Carreno Leandro J; Bueno Susan M; Gonzalez Pablo A;
Mora Jorge E; Quezada Sergio A; Kalergis Alexis M (Reprint)
AUTHOR ADDRESS: Pontificia Univ Catolica Chile, Fac Ciencias Biol, Dept
Genet Mol and Microbiol, Alameda 340, Santiago, Chile**Chile
AUTHOR E-MAIL ADDRESS: akalergis@bio.puc.cl
JOURNAL: Infection and Immunity 74 (11): p6438-6448 NOV 2006 2006
ISSN: 0019-9567
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Dendritic cells (DCs) constitute the link between innate and adaptive immunity by directly recognizing pathogen-associated molecular
patterns (PAMPs) in bacteria and by presenting bacterial antigens to T
cells. Recognition of PAMPs renders DCs as professional antigen-presenting cells able to prime naive T cells and initiate adaptive immunity against bacteria. Therefore, interfering with DC function would promote bacterial survival and dissemination. Understanding the molecular mechanisms that have evolved in virulent bacteria to evade activation of adaptive immunity requires the characterization of virulence factors that interfere with DC function.

Salmonella enterica serovar Typhimurium, the causative agent of typhoid-like disease in the mouse, can prevent antigen presentation to T cells by avoiding lysosomal degradation in DCs. Here, we show that this feature of virulent Salmonella applies in vivo to prevent activation of adaptive immunity. In addition, this attribute of virulent Salmonella requires functional expression of a type three secretion system (ITSS) and effector proteins encoded within the Salmonella pathogenicity island 2 (SPI-2). In contrast to wild-type virulent Salmonella, mutant strains carrying specific deletions of SPI-2 genes encoding TTSS components or effectors proteins are targeted to lysosomes and are no longer able to prevent DCs from activating T cells in vitro or in vivo. SPI-2 mutant strains are attenuated in vivo, showing reduced tissue colonization and enhanced T-cell activation, which confers protection against a challenge with wild-type virulent Salmonella. Our data suggest that impairment of DC function by the activity of SPI-2 gene products is crucial for Salmonella pathogenesis.

3/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

19019187 BIOSIS NO.: 200600364582
Oral vaccine delivery by Salmonella Typhimurium
AUTHOR: Gahan M E (Reprint); Webster D E; Wesselingh S L; Finlay B B; Strugnell R A
AUTHOR ADDRESS: Burnet Inst, Childrens Vaccine Grp, Melbourne, Vic, Australia**Australia
JOURNAL: Tissue Antigens 66 (5): p422-423 NOV 2005 2005
CONFERENCE/MEETING: 35th Annual Scientific Meeting of the Australasian-Society-for-Immunology/14th International HLA and Immunogenetics Workshops Melbourne, AUSTRALIA November 29 -December 02, 2005; 20051129
SPONSOR: Australasian Soc Immunol
ISSN: 0001-2815
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

3/7/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18979823 BIOSIS NO.: 200600325218

Immune response induced by Salmonella typhimurium defective in ppGpp synthesis
AUTHOR: Na Hee Sam (Reprint); Kim Hyun Ju; Lee Hyun-Chul; Hong Yeongjin;
Rhee Joon Haeng; Choy Hyon E
AUTHOR ADDRESS: Chonnam Natl Univ, Sch Med, Dept Microbiol, Kwangju 501746,
South Korea**South Korea
AUTHOR E-MAIL ADDRESS: hyonchoy@chonnam.ac.kr
JOURNAL: Vaccine 24 (12): p2027-2034 MAR 15 2006 2006
ISSN: 0264-410X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Systemic infection by Salmonella typhimurium requires coordinated expression of virulence genes found primarily in Salmonella Pathogenecity Islands (SPIs). We have previously reported that the intracellular signal that induces these virulence genes is a stringent signal molecule, ppGpp [Song et al. J Biol Chem 2003;279:34183]. In this study, we found that relA and spoT double mutant Salmonella (Delta ppGpp strain), which is defective in ppGpp synthesis, was virtually avirulent in BALB/c mice. Subsequently, the live vaccine potential of the avirulent Delta ppGpp Salmonella strain was determined. A single immunization with live Delta ppGpp Salmonella efficiently protected mice from challenge with wild-type Salmonella at a dose 10(6)-fold above the LD50 30 days after immunization. Various assays revealed that immunization of mice with the Delta ppGpp strain elicited both systemic and mucosal antibody responses, in addition to cell-mediated immunity.

3/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18973841 BIOSIS NO.: 200600319236
Cloning of a functional Salmonella SPI-1 type III secretion system and development of a method to create mutations and epitope fusions in the cloned genes
AUTHOR: Wilson James W (Reprint); Nickerson Cheryl A
AUTHOR ADDRESS: Tulane Univ, Hlth Sci Ctr, Program Mol Pathogenesis and Immun, Dept Microbiol and Immunol, 1430 Tulane Ave,Room 5728, New Orleans, LA 70112 USA**USA
AUTHOR E-MAIL ADDRESS: jwilson4@tulane.edu
JOURNAL: Journal of Biotechnology 122 (2): p147-160 MAR 23 2006 2006

ISSN: 0168-1656
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Bacterial type III secretion systems have significant potential to be harnessed for beneficial purposes including vaccine development, anti-cancer therapies, strategies to counteract harmful bacteria-host interactions, and evolutionary studies. The ability to clone and manipulate type III secretion systerns would allow researchers to perform novel experiments that would progress the biotechnological development of the potentially positive uses of these systems. Here, we report the cloning of the entire Salmonella pathogenicity island 1 (SPI-1) type III secretion system on a single DNA fragment that is contained on a self-transmissible plasmid vector for convenient transfer to alternate hosts. We demonstrate that the cloned SPI-1 type III system is functional for secretion and translocation via complementation of an S. typhinutrium Delta SPI-1 strain. We also present a convenient method to construct mutations and epitope fusions in the cloned type III genes and demonstrate that the engineered substrate protein fusions are recognized by the cloned type III system. We transferred the cloned SPI-1 type III system into bacterial strains of different genera and found that there is a SPI-1 gene expression defect in these strains. The results describe a novel strategy for cloning and manipulation of bacterial secretion system gene clusters and provide a foundation for future studies to develop the beneficial uses of cloned type III secretion systems. (c) 2005 Elsevier B.V. All rights reserved.

3/7/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18889641 BIOSIS NO.: 200600235036
Evaluation of Salmonella enterica serovar Typhi (Ty2 aroC-ssaV-) M01ZH09, with a defined mutation in the Salmonella pathogenicity island 2, as a live, oral typhoid vaccine in human volunteers

AUTHOR: Kirkpatrick B D (Reprint); McKenzie Robin; O'Neill J Patrick;
Larsson Catherine J; Bourgeois A Louis; Shimko Janet; Bentley Matthew;
Makin Jill; Chatfield Steve; Hindle Zoe; Fidler Christine; Robinson Brad
E; Ventrone Cassandra H; Bansal Nivedita; Carpenter Colleen M; Kutzko
Deborah; Hamlet Sandra; LaPointe Casey; Taylor David N
AUTHOR ADDRESS: Univ Vermont, Coll Med, Dept Med, Infect Dis Unit, 95
Carrigan Dr,110 Stafford Bldg, Burlington, VT 05405 USA**USA
AUTHOR E-MAIL ADDRESS: beth.kirkpatrick@uvm.edu
JOURNAL: Vaccine 24 (2): p116-123 JAN 12 2006 2006
ISSN: 0264-410X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Salmonella enterica serovar Typhi strains with mutations in
the Salmonella pathogenicity island-2 (SPI-2) may
represent an effective strategy for human vaccine development, and
a vectoring system for heterologous antigens. S. Typhi (Ty2 aroC-ssaV-)
M01ZH09 is an attenuated, live, oral typhoid vaccine
harboring defined deletion mutations in ssaV, which encodes an integral
component in the SPI-2 type HI secretion system (TTSS), as well as a
mutation in an aromatic biosynthetic pathway needed for bacterial growth
in vivo (aroC). SPI-2 mutant vaccines have yet to be evaluated in a
large, randomized human trial. A simplified or single-oral dose oral
typhoid vaccine using the SPI-2 strategy would offer significant
advantages over the currently licensed typhoid vaccines. We
performed a double-blinded, placebo-controlled, dose-escalating clinical
trial in 60 healthy adult volunteers to determine the tolerability and
immunogenicity of a single dose of M01ZH09. Three groups of 20 healthy
adult volunteers were enrolled; 16 in each group received a single oral
dose of the freeze-dried vaccine at 5 x 107, 5 x 108 or 5 x 109 CFU
in a bicarbonate buffer. Four volunteers in each cohort received placebo
in the same buffer. Adverse events were infrequent and not statistically
different between vaccine and placebo recipients, although two
subjects in the mid-range dose and three subjects in the highest dose had
temperature measurements > 37.5 degrees C. No blood or urine cultures
were positive for M01ZH09, and fecal shedding was brief. The immune
response was dose-related; the highest vaccine dose (5 x 109 CFU)

was the most immunogenic. All tested subjects receiving the highest dose had a significant ASC response (mean 118 spots/10(6) cells). A >= 4-fold increase in antibody titer for S. Typhi LPS or flagellin was detected in 75% of volunteers in the highest-dose cohort by day 28. The SPI-2 mutant vaccine, M01ZH09, is a promising typhoid vaccine candidate and deserves further study as a vectoring system for heterologous vaccine antigens.

3/7/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18806788 BIOSIS NO.: 200600152183
Identification of Salmonella gallinarum virulence genes in a chicken infection model using PCR-based signature-tagged mutagenesis
AUTHOR: Shah Devendra H; Lee Mi-jin; Park Jin-ho; Lee John-hwa; Eo Seong-kug; Kwon Jung-thek; Chae Joon-seok (Reprint)
AUTHOR ADDRESS: Chonbuk Natl Univ, Biosafety Res Inst, Jeonju 561756, South Korea**South Korea
AUTHOR E-MAIL ADDRESS: jschae@chonbuk.ac.kr
JOURNAL: Microbiology (Reading) 151 (Part 12): p3957-3968 DEC 2005 2005
ISSN: 1350-0872
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Salmonella gallinarum (SG) is a non-motile host-adapted salmonella that causes fowl typhoid, a severe systemic disease responsible for significant economic losses to the poultry industry worldwide. This study describes the application of a PCR-basecl signature-tagged mutagenesis system to identify in vivo-essential genes of SG. Ninety-six pools representing 1152 SG mutants were screened in a natural-host chicken infection model. Twenty presumptive attenuated mutants were identified and examined further. The identity of the disrupted gene in each mutant was determined by cloning of the DNA sequences adjacent to the transposon, followed by sequencing and comparison with the bacterial genome database. In vitro and in vivo competition indices were determined for each identified mutant and a total of 18 unique, attenuating gene disruptions were identified. These mutations represented six broad genomic classes: Salmonella pathogenicity island-1 (SPI-1), SPI-2, SPI-10, SPI-13, SPI-14 and non-SPI-encoded virulence genes. SPI-1 3 and SPI-14 are newly identified and designated in this study. Most of the genes identified in

this study were not previously believed or known to play a role in the pathogenesis of SG infection in chickens. Each STM identified mutant showed competitiveness and/or virulence defects, confirmed by in vitro and in vivo assays, and challenge tests. This study should contribute to a better understanding of the pathogenic mechanisms involved in progression of disease caused by SG, and identification of novel live vaccine candidates and new potential antibiotic targets.

3/7/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18763986 BIOSIS NO.: 200600109381
Negative regulation of Salmonella pathogenicity island 2 is required for contextual control of virulence during typhoid
AUTHOR: Coombes Brian K; Wickham Mark E; Lowden Michael J; Brown Nat F; Finlay B Brett (Reprint)
AUTHOR ADDRESS: Univ British Columbia, Michael Smith Labs, Vancouver, BC V6T 1Z4, Canada**Canada
AUTHOR E-MAIL ADDRESS: bfinlay@interchange.ubc.ca
JOURNAL: Proceedings of the National Academy of Sciences of the United States of America 102 (48): p17460-17465 NOV 29 2005 2005
ISSN: 0027-8424
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Salmonella enterica relies on a type III secretion system encoded in Salmonella pathogenicity island-2 (SPI-2) to survive and replicate within macrophages at systemic sites during typhoid. SPI-2 virulence is induced upon entry into macrophages, but the mechanisms of SPI-2 gene control in vivo remain unclear, particularly with regard to negative regulators that control the contextual activation of SPI-2. Here, we identified and characterized YdgT as a negative modulator of the SPI-2 pathogenicity island and established that this negative regulation is central to systemic pathogenesis because ydgT mutants overexpressing typhoid virulence genes were ultimately attenuated during infection. ydgT mutants displayed a biphasic virulence phenotype during in vivo competitive infections that consisted of an early "gain-of-virulence" dependent on SPI-2 activation, followed

by attenuation later in infection indicating that proper contextual regulation of SPI-2 by YdgT is necessary for full virulence during systemic colonization. These data suggest that overexpression of virulence-associated type III secretion genes can have an adverse effect on bacterial pathogenesis in vivo.

3/7/9 (Item 9 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18720030 BIOSIS NO.: 200600065425
SlyA regulates function of Salmonella pathogenicity island 2 (SPI-2) and expression of SPI-2-associated genes
AUTHOR: Linehan Sheena A; Rytkonen Anne; Yu Xiu-Jun; Liu Mei; Holden David W (Reprint)
AUTHOR ADDRESS: Univ London Imperial Coll Sci Technol and Med, Dept Infect Dis, Ctr Mol Microbiol and Infect, Flowers Bldg,Armstrong Rd, London SW7 2AZ, UK**UK
AUTHOR E-MAIL ADDRESS: d.holden@imperial.ac.uk
JOURNAL: Infection and Immunity 73 (7): p4354-4362 JUL 2005 2005
ISSN: 0019-9567
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: During the systemic phase of murine infection with Salmonella enterica serovar Typhimurium, bacterial virulence is correlated with the ability to grow and survive within host macrophages. Salmonella pathogenicity island 2 (SPI-2), encoding a type three secretion system, has emerged as an important contributor to Salmonella intracellular growth. SPI-2 mutants have been proposed to be more accessible than wild-type Salmonella to oxyradicals generated by the NADPH phagocyte oxidase. We performed mixed infections of mice to investigate the relationship between SPI-2 and SlyA, a transcriptional regulator that confers resistance to oxyradicals. In mixed-infection experiments, the SPI-2 null mutant was severely attenuated in virulence, whereas slyA mutants were only mildly attenuated. Surprisingly, further experiments indicated that the function of SPI-2 was partially dependent on slyA. The intracellular behavior of a slyA mutant in infected cells was consistent with inefficient SPI-2 expression, as formation of Salmonella-induced filaments and the intracellular F-actin meshwork, features that depend on SPI-2, were present at abnormally low frequencies. Furthermore, the

translocated levels of the SPI-2 effector Ssej were severely reduced in a strain carrying a mutation in slyA. We used flow cytometry to investigate the role of SlyA in expression of green fluorescent protein (GFP) from transcriptional fusions with promoters of either of two other SPI-2 effector genes, sijB and sifA. The slyA mutant exhibited reduced GFP expression from both promoters. Combining mutations in slyA and other regulators of SPI-2 indicated that SlyA acts through the SsrAB two-component regulatory system. SlyA exhibits partial functional redundancy with OmpR-EnvZ and contributes to the transcriptional response to low osmolarity and the absence of calcium, two environmental stimuli that promote SPI-2 gene expression.

3/7/10 (Item 10 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18712645 BIOSIS NO.: 200600058040
Resolvase-in vivo expression technology analysis of the Salmonella enterica serovar typhimurium PhoP and PmrA regulons in BALB/c mice
AUTHOR: Merighi Massimo; Ellermeier Craig D; Slauch James M; Gunn John S (Reprint)
AUTHOR ADDRESS: 270 TMRF,420 W 12th Ave, Columbus, OH 43210 USA**USA
AUTHOR E-MAIL ADDRESS: gunn.43@osu.edu
JOURNAL: Journal of Bacteriology 187 (21): p7407-7416 NOV 2005 2005
ISSN: 0021-9193
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Salmonella enterica modulates resistance to antimicrobial peptides in part via covalent modifications of the lipopolysaccharide (LPS). The two-component systems PhoP/PhoQ and PmrA/PmrB are activated during infection and regulate several genes involved in LPS modifications by responding to signals such as pH, iron, magnesium, and antimicrobial peptides. A recombination-based in vivo expression technology approach was adopted to analyze the spatial-temporal patterns of in vivo expression of genes of the PhoP and PmrA regulons and to identify the in vivo signals modulating their transcription. In vitro, we showed PhoP-

and/or PmrA-dependent induction of pmrH (LPS aminoarabinose modification operon) by acidic pH, low levels of magnesium, or high levels of Fe(III). Upregullation in cultured J774A.1 macrophages was shown for pmrH, pagP (LPS palmitate addition), and ssaB (pathogenicity island II secretion) but not for prgH (pathogenicity island I secretion). Increased levels of pmrH, phoP, and prgH transcription but not ssaB were observed in bacteria isolated from the lumen of the distal ileum. Bacteria isolated from spleens of orally inoculated mice showed no further induction of prgH but had the highest expression of pmrH, pagP, and ssaB. In vivo induction of pmrH was fully dependent on pmrA and phoP, and buffering stomach acidity, iron chelation, or low-iron diets did not affect the expression of pmrH in the intestinal lumen. The observation of pmrH and pagP expression in the intestine refutes the paradigm of PhoP/PhoQ and PmrA/PmrB in vivo expression as solely intracellularly induced and supports previous data demonstrating peroral virulence attenuation of pmrH mutants.
? ds

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                OR LEUX OR PATHOGENICITY(W)ISLAND)
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S3        139   S2 NOT PY>2006
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             234  LEUX
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      S4       0  S3 AND (LEUX OR TRNA5LEU)
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          135342  TRNA
      S5       1  S3 AND TRNA
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 5/7/1     (Item 1 from file: 34)
DIALOG(R)File  34:SciSearch(R) Cited Ref Sci
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12588742   Genuine Article#: 802YL   Number of References: 126
Title: Plasticity of bacterial genomes: Pathogenicity islands and the Locus
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of Enterocyte Effacement (LEE)
Author(s): Kirsch P; Jores J; Wieler LH (REPRINT)
Corporate Source: Free Univ Berlin,Fachbereich Vet Med, Inst Mikrobiol &
Tierseuchen,Postfach 04 02 25/D-10061 Berlin//Germany/ (REPRINT); Free
Univ Berlin,Fachbereich Vet Med, Inst Mikrobiol & Tierseuchen,D-10061
Berlin//Germany/; Univ Ulm,Tierforschungszentrum,D-89069 Ulm//Germany/
Journal: BERLINER UND MUNCHENER TIERARZTLICHE WOCHENSCHRIFT, 2004, V117,
N3-4 (MAR-APR), P116-129
ISSN: 0005-9366 Publication date: 20040300
Publisher: SCHLUETERSCHE GMBH & CO K G VERLAG UND DRUCKEREI,
HANS-BOCKLER-ALLEE 7, 30173 HANNOVER, GERMANY
Language: German Document Type: REVIEW
Abstract: Many bacterial virulence attributes, like toxins, adhesins, invasins, iron uptake systems, are encoded within specific regions of the bacterial genome. These in size varying regions are termed pathogenicity islands (PAIs) since they confer pathogenic properties to the respective micro-organism. Per definition PAIs are exclusively found in pathogenic strains and are often inserted near transfer-RNA genes. Nevertheless, non-pathogenic bacteria also possess foreign DNA elements that confer advantageous features, leading to improved fitness. These additional DNA elements as well as PAIs are termed genomic islands and were acquired during bacterial evolution. Significant G+C content deviation in pathogenicity islands with respect to the rest of the genome, the presence of direct repeat sequences at the flanking regions, the presence of integrase gene determinants as other mobility features, the particular insertion site (tRNA gene) as well as the observed genetic instability suggests that pathogenicity islands were acquired by horizontal gene transfer PAIs are the fascinating proof of the plasticity of bacterial genomes. PAIs were originally described in human pathogenic Escherichia (E.) coli strains. In the meantime PAIs have been found in various pathogenic bacteria of humans, animals and even plants. The Locus of Enterocyte Effacement (LEE) is one particular widely distributed PAI of E coli. In addition, it also confers pathogenicity to the related species

Citrobacter (C.) rodentium and Escherichia (E.) alvei. The LEE is an important virulence feature of several animal pathogens. It is an obligate PAI of all animal and human enteropathogenic E coli (EPEC), and most enterohaemorrhegic E coli (EHEC) also harbor the LEE.

The LEE encodes a type III secretion system, an adhesion (intimin) that mediates the intimate contact between the bacterium and the epithelial cell, as well as various proteins which are secreted via the type III secretion system. The LEE encoded virulence features are responsible for the formation of so called attaching and effacing (AE) lesions in the intestinal epithelium. Due to its wide distribution in animal pathogens, LEE encoded antigens are suitable vaccine antigens. Acquisition and structure of the LEE pathogenicity island is the crucial point of numerous investigations. However, the evolution of the LEE, its origin and further spread in E coli, are far from being resolved.

```
? s s3 and supp
             139  S3
            3669  SUPP
      S6       0  S3 AND SUPP
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Set     Items   Description
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                OR LEUX OR PATHOGENICITY(W)ISLAND)
S2        174   RD S1  (unique items)
S3        139   S2 NOT PY>2006
S4          0   S3 AND (LEUX OR TRNA5LEU)
S5          1   S3 AND TRNA
S6          0   S3 AND SUPP
? s (attenuat? or avirulent or vaccin?) and (typhi or dublin or
typhimurium) and (PAI or leuX or pathogenicity(w)island)
          999613  ATTENUAT?
           30122  AVIRULENT
         1153992  VACCIN?
           39490  TYPHI
           21480  DUBLIN
          209295  TYPHIMURIUM
           51654  PAI
             234  LEUX
          302725  PATHOGENICITY
          466932  ISLAND
           10789  PATHOGENICITY(W)ISLAND
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DUBLIN
                  OR TYPHIMURIUM) AND (PAI OR LEUX OR
                  PATHOGENICITY(W)ISLAND)
? rd s7

>>>Duplicate detection is not supported for File 393.

>>>Records from unsupported files will be retained in the RD set.
      S8     163  RD S7  (unique items)
? s s8 and (leux or trna or supp)
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             234  LEUX
          135342  TRNA
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      S9       1  S8 AND (LEUX OR TRNA OR SUPP)
? t s9/7/1
>>>Format 7 is not valid in file 143

 9/7/1     (Item 1 from file: 34)
DIALOG(R)File  34:SciSearch(R) Cited Ref Sci
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12588742   Genuine Article#: 802YL   Number of References: 126
Title: Plasticity of bacterial genomes: Pathogenicity islands and the
Locus
    of Enterocyte Effacement (LEE)
Author(s): Kirsch P; Jores J; Wieler LH  (REPRINT)
Corporate Source: Free Univ Berlin,Fachbereich Vet Med, Inst
Mikrobiol &
    Tierseuchen,Postfach 04 02 25/D-10061 Berlin//Germany/ (REPRINT);
Free
    Univ Berlin,Fachbereich Vet Med, Inst Mikrobiol &
Tierseuchen,D-10061
    Berlin//Germany/; Univ Ulm,Tierforschungszentrum,D-89069
Ulm//Germany/
Journal: BERLINER UND MUNCHENER TIERARZTLICHE WOCHENSCHRIFT, 2004,
V117,
    N3-4 (MAR-APR), P116-129
ISSN: 0005-9366   Publication date: 20040300
Publisher: SCHLUETERSCHE GMBH & CO K G VERLAG UND DRUCKEREI,
    HANS-BOCKLER-ALLEE 7, 30173 HANNOVER, GERMANY
Language: German   Document Type: REVIEW
Abstract: Many bacterial virulence attributes, like toxins, adhesins,
    invasins, iron uptake systems, are encoded within specific
regions of
    the bacterial genome. These in size varying regions are termed
    pathogenicity islands (PAIs) since they confer pathogenic
properties to
    the respective micro-organism. Per definition PAIs are exclusively
    found in pathogenic strains and are often inserted near
transfer-RNA
```

genes. Nevertheless, non-pathogenic bacteria also possess foreign DNA elements that confer advantageous features, leading to improved fitness. These additional DNA elements as well as PAIs are termed genomic islands and were acquired during bacterial evolution. Significant G+C content deviation in pathogenicity islands with respect to the rest of the genome, the presence of direct repeat sequences at the flanking regions, the presence of integrase gene determinants as other mobility features, the particular insertion site (tRNA gene) as well as the observed genetic instability suggests that pathogenicity islands were acquired by horizontal gene transfer PAIs are the fascinating proof of the plasticity of bacterial genomes. PAIs were originally described in human pathogenic Escherichia (E.) coli strains. In the meantime PAIs have been found in various pathogenic bacteria of humans, animals and even plants. The Locus of Enterocyte Effacement (LEE) is one particular widely distributed PAI of E coli. In addition, it also confers pathogenicity to the related species Citrobacter (C.) rodentium and Escherichia (E.) alvei. The LEE is an important virulence feature of several animal pathogens. It is an obligate PAI of all animal and human enteropathogenic E coli (EPEC), and most enterohaemorrhegic E coli (EHEC) also harbor the LEE. The LEE encodes a type III secretion system, an adhesion (intimin) that mediates the intimate contact between the bacterium and the epithelial cell, as well as various proteins which are secreted via the type III secretion system. The LEE encoded virulence features are responsible for the formation of so called attaching and effacing (AE) lesions in the intestinal epithelium. Due to its wide distribution in animal pathogens, LEE encoded antigens are suitable vaccine antigens. Acquisition and structure of the LEE pathogenicity island is the crucial point of numerous investigations. However, the evolution of the LEE, its origin and further spread in E coli, are far from being resolved.

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               OR LEUX OR PATHOGENICITY(W)ISLAND)
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S6          0   S3 AND SUPP
S7        462   (ATTENUAT? OR AVIRULENT OR VACCIN?) AND (TYPHI OR
DUBLIN OR
               TYPHIMURIUM) AND (PAI OR LEUX OR PATHOGENICITY(W)ISLAND)
S8        163   RD S7  (unique items)
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? e au=cohen, paul s.

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E2       30  AU=COHEN, PAUL S
E3       77 *AU=COHEN, PAUL S.
E4        2  AU=COHEN, PAUL SHEA
E5        3  AU=COHEN, PAUL SIDNEY
E6       11  AU=COHEN, PAULA E
E7       27  AU=COHEN, PAULA E.
E8        4  AU=COHEN, PAZIT Y
E9        2  AU=COHEN, PAZIT Y.
E10       2  AU=COHEN, PD
E11      15  AU=COHEN, PE
E12       1  AU=COHEN, PEDRO

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PLEASE ENTER A COMMAND OR BE LOGGED OFF IN 5 MINUTES
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               OR LEUX OR PATHOGENICITY(W)ISLAND)
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S3        139   S2 NOT PY>2006
S4          0   S3 AND (LEUX OR TRNA5LEU)
S5          1   S3 AND TRNA
S6          0   S3 AND SUPP
S7        462   (ATTENUAT? OR AVIRULENT OR VACCIN?) AND (TYPHI OR
DUBLIN OR
               TYPHIMURIUM) AND (PAI OR LEUX OR PATHOGENICITY(W)ISLAND)
S8        163   RD S7  (unique items)
S9          1   S8 AND (LEUX OR TRNA OR SUPP)
? e au=cohen, paul s.

Ref   Items  Index-term
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E3        77 *AU=COHEN, PAUL S.
E4         2  AU=COHEN, PAUL SHEA
E5         3  AU=COHEN, PAUL SIDNEY
E6        11  AU=COHEN, PAULA E
E7        27  AU=COHEN, PAULA E.
E8         4  AU=COHEN, PAZIT Y
E9         2  AU=COHEN, PAZIT Y.
E10        2  AU=COHEN, PD
E11       15  AU=COHEN, PE
E12        1  AU=COHEN, PEDRO

          Enter P or PAGE for more
? s e1-e5
               1  AU=COHEN, PAUL R
              30  AU=COHEN, PAUL S
              77  AU=COHEN, PAUL S.
               2  AU=COHEN, PAUL SHEA
               3  AU=COHEN, PAUL SIDNEY
     S10     113  E1-E5
? e au=cohen, p.s.

Ref   Items  Index-term
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E2        8  AU=COHEN, P.R.
E3        3 *AU=COHEN, P.S.
E4        1  AU=COHEN, P.T.W.
E5        7  AU=COHEN, P*
E6       31  AU=COHEN, PA
E7        1  AU=COHEN, PA*
E8        1  AU=COHEN, PAM
E9        8  AU=COHEN, PAMELA
E10       1  AU=COHEN, PAMELA E
E11       1  AU=COHEN, PAMELA E.
E12       3  AU=COHEN, PAMELA S

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E1      285  AU=COHEN, P
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E3        0 *AU=COHEN, P S
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E5        2  AU=COHEN, P. (ED.)
E6        1  AU=COHEN, P. (LGH (CANADA))
E7       25  AU=COHEN, P. A.
E8        2  AU=COHEN, P. ARYEH
E9       12  AU=COHEN, P. B.
E10       1  AU=COHEN, P. BEAZLEY
E11       2  AU=COHEN, P. C.
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E12       5  AU=COHEN, P. D.

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E2      208  AU=COHEN, P. R.
E3       46 *AU=COHEN, P. S.
E4       13  AU=COHEN, P. T.
E5       26  AU=COHEN, P. T. W.
E6        1  AU=COHEN, P. T. WADE
E7        1  AU=COHEN, P. Y.
E8        3  AU=COHEN, P.A.
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E10       1  AU=COHEN, P.E.
E11       1  AU=COHEN, P.H.
E12      35  AU=COHEN, P.I.

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     S11      46  AU='COHEN, P. S.'
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      Display 11/2/1     (Item 1 from file: 6)
DIALOG(R)File   6:NTIS
(c) 2008 NTIS, Intl Cpyrght All Rights Res. All rts. reserv.

1882914  NTIS Accession Number: AD-A233 255/9
  Growth  of  Salmonella  typhimurium  SL5319  and Escherichia coli
F-18 in
Mouse  Cecal  Mucus:  Role  of  Peptides and Iron. (Reannouncement
with New
Availability Information)
  (Journal article)
  Franklin, D. P. ; Laux, D. C. ; Williams, T. J. ; Falk, M. C. ;
Cohen,
P. S.
  Naval Medical Research Inst., Bethesda, MD.
  Corp. Source Codes: 019861000; 249650
  Report Number: NMRI-90-126
  1990   12p
  Languages: English   Document Type: Journal article
  Journal Announcement: GRAI9517
  Pub.  in FEMS Microbiology Ecology, v74 p229-240 1990. Order this
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(703)605-6000
(other     countries);    fax    at    (703)321-8547;    and    email
   at

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? ds
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Set     Items   Description
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AND (PAI
               OR LEUX OR PATHOGENICITY(W)ISLAND)
S2        174   RD S1  (unique items)
S3        139   S2 NOT PY>2006
S4          0   S3 AND (LEUX OR TRNA5LEU)
S5          1   S3 AND TRNA
S6          0   S3 AND SUPP
S7        462   (ATTENUAT? OR AVIRULENT OR VACCIN?) AND (TYPHI OR
DUBLIN OR
               TYPHIMURIUM) AND (PAI OR LEUX OR PATHOGENICITY(W)ISLAND)
S8        163   RD S7  (unique items)
S9          1   S8 AND (LEUX OR TRNA OR SUPP)
S10       113   E1-E5
S11        46   AU='COHEN, P. S.'
? s s10 or s11
             113  S10
              46  S11
     S12     159  S10 OR S11
? s s12 and (leux or trna or supp)
             159  S12
             234  LEUX
          135342  TRNA
            3669  SUPP
     S13       3  S12 AND (LEUX OR TRNA OR SUPP)
? t s13/7/all
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 13/7/1     (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
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  129038689    CA: 129(4)38689b    JOURNAL
  The leuX-encoded tRNA5Leu but not the pathogenicity islands I and II
  influences the survival of the uropathogenic Escherichia coli
strain 536
  in CD-1 mouse bladder mucus in the stationary phase
  AUTHOR(S): Dobrindt, Ulrich; Cohen, Paul S.; Utley, Maryjane;
Muhldorfer,
Inge; Hacker, Jorg
  LOCATION: Institut fur Molekulare Infektionsbiologie, Universitat
Wurzburg, Wurzburg, Germany, 97070
  JOURNAL: FEMS Microbiol. Lett.  DATE: 1998  VOLUME: 162  NUMBER: 1
  PAGES: 135-141  CODEN: FMLED7  ISSN: 0378-1097  LANGUAGE: English
  PUBLISHER: Elsevier Science B.V.
  SECTION:
    CA210006 MICROBIAL, ALGAL, AND FUNGAL BIOCHEMISTRY
    CA214XXX Mammalian Pathological Biochemistry
  IDENTIFIERS: gene leuX pathogenicity Escherichia bladder
  DESCRIPTORS:
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Bladder... Escherichia coli... Mouse... Mucus...
Virulence(microbial)...
    gene leuX-encoded tRNA5Leu influences survival of uropathogenic
    Escherichia coli in mouse bladder mucus
tRNA...
    leucine-specific; gene leuX-encoded tRNA5Leu influences survival
of
    uropathogenic Escherichia coli in mouse bladder mucus
Genes(microbial)...
    leuX; gene leuX-encoded tRNA5Leu influences survival of
uropathogenic
    Escherichia coli in mouse bladder mucus

 13/7/2   (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

  122211009   CA: 122(17)211009k   JOURNAL
  Role of leuX in Escherichia coli colonization of the
streptomycin-treated
  mouse large intestine
  AUTHOR(S): Newman, Joseph V.; Kolter, Roberto; Laux, David C.;
Cohen,
Paul S.
  LOCATION: Department of Biochemistry, Microbiology and Molecular
Genetics
, University of Rhode Island, Kingston, RI, 02881, USA
  JOURNAL: Microb. Pathog. DATE: 1994 VOLUME: 17 NUMBER: 5 PAGES:
301-11 CODEN: MIPAEV ISSN: 0882-4010 LANGUAGE: English
  SECTION:
    CA214003 Mammalian Pathological Biochemistry
    CA210XXX Microbial Biochemistry
  IDENTIFIERS: gene leuX Escherichia colonization intestine
  DESCRIPTORS:
Escherichia coli...
    colonization of streptomycin-treated mouse large intestine
mediated by
    Escherichia coli mediated by leuX gene encoding leucine-specific
tRNA
Gene,microbial, leuX...
    Escherichia coli colonization of the streptomycin-treated mouse
large
    intestine mediated by
Ribonucleic acids,transfer, leucine-specific CAA...
    Escherichia coli colonization of the streptomycin-treated mouse
large
    intestine mediated by leuX gene encoding
Intestine,disease, large...
    infection; Escherichia coli colonization of the
streptomycin-treated
    mouse large intestine mediated by leuX gene encoding
leucine-specific

tRNA
Microorganism growth,stationary phase...
leuX gene expression required for survival in stationary phase of Escherichia coli in streptomycin-treated mouse large intestine

13/7/3 (Item 3 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

121251015 CA: 121(21)251015v JOURNAL
Stimulation of Escherichia coli F-18Col- Type-1 fimbriae synthesis by
leuX
AUTHOR(S): Newman, Joseph V.; Burghoff, Robert L.; Pallesen, Lars; Krogfelt, Karen A.; Kristensen, Claus S.; Laux, David C.; Cohen, Paul S.
LOCATION: Department of Biochemistry, Microbiology, and Molecular Genetics, University of Rhode Island, Kingston, RI, 02881, USA
JOURNAL: FEMS Microbiol. Lett. DATE: 1994 VOLUME: 122 NUMBER: 3
PAGES: 281-8 CODEN: FMLED7 ISSN: 0378-1097 LANGUAGE: English
SECTION:
CA210004 Microbial Biochemistry
CA203XXX Biochemical Genetics
IDENTIFIERS: gene leuX type 1 fimbriae Escherichia
DESCRIPTORS:
Gene,microbial, leuX...
for leucineCAA-specific tRNA; type 1 fimbriae synthesis in Escherichia
coli is stimulated by gene leuX
Ribonucleic acids,transfer, leucine-specific CAA...
gene leuX-encoded; type 1 fimbriae synthesis in Escherichia coli is
stimulated by gene leuX
Proteins,specific or class, 26,000-mol.-weight...
neg. regulator; gene leuX expression regulation in Escherichia coli
Proteins,specific or class, 22,000-mol.-weight...
pos. regulator; gene leuX expression regulation in Escherichia coli
Escherichia coli... Pili,type 1...
type 1 fimbriae synthesis in Escherichia coli is stimulated by gene
leuX
? s (leux or trna5leu or supp)
234 LEUX
38 TRNA5LEU
3669 SUPP
S14 3901 (LEUX OR TRNA5LEU OR SUPP)
? rd s14

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S5          1   S3 AND TRNA
S6          0   S3 AND SUPP
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DUBLIN OR
              TYPHIMURIUM) AND (PAI OR LEUX OR PATHOGENICITY(W)ISLAND)
S8        163   RD S7  (unique items)
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S10       113   E1-E5
S11        46   AU='COHEN, P. S.'
S12       159   S10 OR S11
S13         3   S12 AND (LEUX OR TRNA OR SUPP)
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          915106  VARIANT?
            1462  MUTEIN
           30122  AVIRULENT
          999613  ATTENUAT?
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 17/7/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
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18719996   BIOSIS NO.: 200600065391
Multiple insertional events, restricted by the genetic background,
have led
  to acquisition of pathogenicity island IIJ96-like domains among
  Escherichia coli strains of different clinical origins
AUTHOR: Bidet Philippe; Bonacorsi Stephane; Clermont Olivier; De
Montille
  Caroline; Brahimi Naima; Bingen Edouard (Reprint)
AUTHOR ADDRESS: Hop Robert Debre, Microbiol Serv, 48 Bd Serurier,
F-75395
  Paris 19, France**France
AUTHOR E-MAIL ADDRESS: edouard.bingen@rdb.ap-hop-paris.fr
JOURNAL: Infection and Immunity  73 (7): p4081-4087 JUL 2005 2005
ISSN: 0019-9567
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: We investigated the dissemination of pathogenicity island
(PAI)
  IIJ96-Iike elements (hra, hly, cnfI, and pap) among 455 Escherichia
coli
  isolates from children and adults with urinary tract infection
(UTI),
  neonates with meningitis or colonized healthy neonates, and 74
reference
  strains by means of PCR phylogenetic grouping, ribotyping, and PCR
  analysis of virulence genes. Colocalization of these genes was
documented
  by pulsed-field gel electrophoresis followed by Southern
hybridization
  and long-range PCR (LRPCR) between the hra and the papG alleles.
  Site-specific insertion of the PAI was determined by LRPCR between
hra
  and tRNA flanking sequences. hra, hly, and cnfI were found in 113
  isolates and consistently colocalized, constituting the backbone of
PAI
```

IIJ96-like domains. The prevalence of PAI IIJ96-like domains was significantly higher among UTI isolates than among neonatal meningitis and commensal isolates. These domains were restricted to a few ribotypes of group B2. In contrast to the consistent colocalization of hra, hly, and cnfI, the pap operon was varied: 12% of strains exhibited an allelic exchange of the papG class III allele (papGIII) for the papG class II allele (papGII) (only UTI isolates), and the pap operon was deleted in 23% of strains. No strains harbored papGIII outside the PAI, which appears to be the only source of this allele. PAI IIJ96-like domains were inserted in the vicinities of three different tRNAs-pheU (54%), leuX (29%), and pheV (15%)-depending on the genetic backgrounds and origins of the isolates. Multiple insertional events restricted by the genetic background have thus led to PAI IIJ96 acquisition. Specific genetic backgrounds and insertion sites may have played a role in additional recombination processes for E. coli adaptation to different ecological niches.

17/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17916178 BIOSIS NO.: 200400286935
Inhibition of late airway responses in ovine and murine models of asthma by oral heparin-tetrasaccharide
AUTHOR: Ahmed Tahir (Reprint); Abraham William M
AUTHOR ADDRESS: Pulmonary Division, Mount Sinai Medical Center, 4300 Alton Road, Miam Beach, FL, 33140, USA**USA
AUTHOR E-MAIL ADDRESS: tahmed@msmc.com
JOURNAL: FASEB Journal 18 (4-5): pAbst. 769.10 2004 2004
MEDIUM: e-file
CONFERENCE/MEETING: FASEB Meeting on Experimental Biology: Translating the Genome Washington, District of Columbia, USA April 17-21, 2004; 20040417
SPONSOR: FASEB
ISSN: 0892-6638 _(ISSN print)
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Inhaled heparin-derived oligosaccharides attenuate allergic airway responses in sheep. The anti-allergic activity resides in a tetrasaccharide (Tetra) sequence (J Appl Physiol 88:1721, 2000; AJR CCM 1998; 157:A826). Here, we studied the effects of orally administered Tetra on antigen-induced early (EAR) and late (LAR) airway responses and airway hyperresponsiveness (AHR) in allergic sheep and eosinophil influx in a murine model of asthma. Specific lung resistance (SRL) was measured in 8 allergic sheep before and for 8 h after challenge with Ascaris suum antigen, without and after treatment with oral Tetra (0.06, 0.125 and 0.25 mg/kg). AHR was based on the change in the dose inhaled carbachol which increased SRL by 400% (PD400) before and 24h after antigen challenge. BALB/c mice were sensitized, challenged with aerosolized ovalbumin, following pretreatment with 100 ug oral Tetra or placebo, and eosinophil cell count in BAL was measured 24 h later. In sheep, Tetra at 0.06, 0.125 and 0.25 mg/kg inhibited the EAR by 29%, 67%, and 74%, while LAR was inhibited by 2%, 59% and 72%, respectively. Tetra also inhibited the antigen-induced AHR dose-dependently: PD400 was 46% (% pre-challenge value) in controls as compared to 100% with 0.25 mg/kg Tetra. In mice, Tetra inhibited the antigen induced increase in BAL eosinophils by 51%. These data demonstrate that orally administered heparin-derived Tetra may have therapeutic potential as an anti-inflammatory agent. Supp by Ivax.

17/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17378854 BIOSIS NO.: 200300335597
Swapping Solvent-Exposed Hydrophobic Amino Acids between the Factor VIII and Factor V C2 Domains Causes Reciprocal Changes in Phospholipid Affinities.
AUTHOR: Gilbert Gary E (Reprint); Kaufman Randal J (Reprint); Price Patricia (Reprint); Miao Hongzhi (Reprint); Sirachainan Nongnuch (Reprint); Deng Xuehong (Reprint); Pipe Steven (Reprint)
AUTHOR ADDRESS: Medicine, VA Hospital, Brigham and Women's Hospital,

Harvard Medical School, Boston, MA, USA**USA
JOURNAL: Blood 100 (11): pAbstract No. 997 November 16, 2002 2002
MEDIUM: print
CONFERENCE/MEETING: 44th Annual Meeting of the American Society of Hematology Philadelphia, PA, USA December 06-10, 2002; 20021206
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Factor VIII (fVIII) binds to phospholipid (PL) membranes, von Willebrand factor (vWf) and factor IXa (Gilbert, Thromb. Haemost. 2001 Supp OC1659) via motifs localized to the C2 domain. We have shown that PL binding and vWf binding are mediated by two pairs of hydrophobic residues, each displayed at the tips of b-hairpin turns. The homologous hydrophobic residues in the C2 domain of factor V also contribute to PL binding. We hypothesized that the solvent-exposed hydrophobic residues of the fVIII C2 domain make specific contacts with PL that may explain the different PL binding properties of the two proteins. To test this hypothesis we have prepared fVIII/fV hybrid mutants in which either amino acid(s) of the fVIII C2 domain were changed to the homologous residues of fV (Mutants 1-M/F 2199/2200 W/W, 2-L/L 2251/2252 L/S, and 3-M/F/L 2199/2200/2252 W/W/S) or the complementary fV/fVIII hybrid mutations in which amino acids of the fV C2 domain were changed to the homologous residues of fVIII (Mutants 4 W/W 2063/2064 M/F, 5 L/S 2116/2117 L/L, and 6 W/W/S 2063/2064/2117 M/F/L). All mutants were expressed in COS-1 cells and protein purified by immunoaffinity chromatography or FPLC for fVIII and fV, respectively. fVIII/fV hybrid mutants 1-3 had fVIII specific activities that equaled or exceeded wild-type (WT) fVIII in both 1-stage and 2-stage commercial aPTT assays that contain a large excess of PL. In a PL-limiting Xase assay (sonicated vesicles of PS:PE:PC 4:20:76, 0.15 muM PL) the mutants had 80-95% reduction in specific activity. Phospholipid titration indicated that the apparent phospholipid affinities were higher, lower, and unchanged for mutants 1-3. The maximum catalytic rate with saturating PL and factor IXa was within 25% of WT fVIII for mutants 1-3. The specific activity of the

fV/fVIII hybrids exceeded those of WT fV in a prothrombin time assay with fV deficient plasma. Preliminary data indicate that the relative affinities for PL are > 10-fold higher than WT fV for mutants 5 and 6 which contain the Ser-->Leu change in the second hydrophobic spike. In addition, activity of mutants 5 and 6 is completely suppressed by phospholipid concentrations > 1 muM. Together, these results indicate that Leu 2252 vs. Ser in the second hydrophobic spike enhances PL affinity at least 9-fold for both fVIII and fV. In contrast, the fV Trp-Trp 2163/64 pair, constituting the residues of the 1st hydrophobic spike, appears to confer a somewhat higher affinity than the Met-Phe 2199/00 pair of fVIII. Thus, it appears that fVIII relies particularly on the second hydrophobic spike for high affinity PL binding, whereas fV preferentially requires residues in the first hydrophobic spike.

17/7/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17300103 BIOSIS NO.: 200300258747
Ganglioside-mediated signaling in the control of nerve regeneration.
AUTHOR: Schnaar Ronald L (Reprint); Fredericks Gregory J; Vyas Alka A
AUTHOR ADDRESS: Depts. of Pharmacology and Neuroscience, The Johns Hopkins School of Medicine, 725 N. Wolfe Street, Baltimore, MD, 21205, USA**USA
AUTHOR E-MAIL ADDRESS: schnaar@jhu.edu; gfrederi@mail.jhmi.edu; avays@jhmi.edu
JOURNAL: FASEB Journal 17 (4-5): pAbstract No. 353.2 March 2003 2003
MEDIUM: e-file
CONFERENCE/MEETING: FASEB Meeting on Experimental Biology: Translating the Genome San Diego, CA, USA April 11-15, 2003; 20030411
SPONSOR: FASEB
ISSN: 0892-6638 _(ISSN print)
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Gangliosides are the major sialoglycoconjugates in the brain.
Some ganglioside functions are mediated by binding to a brain lectin, myelin-associated glycoprotein (MAG, Siglec-4), found on myelin membranes

throughout the nervous system. MAG stabilizes axon-myelin interactions and inhibits nerve regeneration after injury. MAG binds to the major brain gangliosides GD1a and GT1b. MAG-mediated inhibition of neurite outgrowth from primary neurons in vitro is attenuated by: (i) neuraminidase; (ii) blocking ganglioside biosynthesis; (iii) genetically modifying gangliosides; and (iv) IgG-class anti-GD1a or anti-GT1b monoclonal antibodies. Furthermore, neurite outgrowth inhibition is mimicked by multivalent clustering of GD1a or GT1b using pre-complexed antibodies.MAG and GD1a clustering both inhibit neurite outgrowth from NG108-15 neuroblastoma-glioma cells. After neuraminidase treatment to convert GD1a to GM1, neither anti-GD1a nor anti-GM1 antibodies inhibited neurite outgrowth. Inhibition via GD1a clustering was mediated by RhoA GTPase. GD1a clustering activated RhoA and treatment with a RhoA inhibitor, C3, blocked neurite outgrowth inhibition. Transfected GFP-RhoA redistributed from the cell body to neurites upon ganglioside clustering and prior to neurite retraction. These data implicate gangliosides as ligands linking MAG binding to neurite outgrowth inhibition via RhoA activation. Supp. by PHS grant NS37096. GJF is a Howard Hughes Predoctoral Fellow.

17/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16891047 BIOSIS NO.: 200200484558
Efficient expression of the alpha-haemolysin determinant in the uropathogenic Escherichia coli strain 536 requires the leuX-encoded tRNA5Leu
AUTHOR: Dobrindt U; Emody L; Gentschev I; Goebel W; Hacker J (Reprint)
AUTHOR ADDRESS: Institut fuer Molekulare Infektionsbiologie, Universitaet Wuerzburg, Roentgenring 11, 97070, Wuerzburg, Germany**Germany
JOURNAL: MGG Molecular Genetics and Genomics 267 (3): p370-379 May, 2002
2002
MEDIUM: print
ISSN: 1617-4615
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The uropathogenic Escherichia coli strain 536 (O6:K15:H31) carries two alpha-haemolysin determinants which are located on different pathogenicity islands (PAI I536 and PAI II536). PAI II536 is associated with the tRNA gene leuX. The leuX-encoded tRNA5Leu is required for the efficient expression of the hly determinants in strain 536. HlyA levels were reduced and secretion of the protein was delayed in the leuX-negative mutant strain 536DELTA102. The lack of a functional tRNA5Leu resulted in a decrease in hly transcript levels in comparison to the wild-type strain. Analysis of several genes whose products are involved in the regulation of hly expression revealed that levels of RfaH and Hha, as well as the corresponding rfaH and hha transcripts, were higher in the leuX-negative background, whereas the expression of tolC and hns was not influenced by the leuX genotype. The analysis of hly transcript levels in hha deletion mutants of the E. coli strains 536 and 536DELTA102 demonstrated that the increase in hha expression is partially responsible for the reduction in hly transcript levels in the leuX-negative background. These results demonstrate that the tRNA5Leu affects the expression of the alpha-haemolysin determinant at different levels in a regulatory cascade, and imply that, in addition to Hha, at least one further, as yet unidentified, regulatory factor must be involved in the regulation of hly transcription in the uropathogenic E. coli strain 536.

16630291 BIOSIS NO.: 200200223802
Effect of COX inhibitors on electrolyte conductances of the frog gastric epithelium
AUTHOR: Carrasquer G (Reprint); Li M (Reprint)
AUTHOR ADDRESS: Dept. of Medicine/Nephrology, Univ. of Lou., Louisville, KY, USA**USA
JOURNAL: Journal of the American Society of Nephrology 11 (Program and Abstract Issue): p26A September, 2000 2000
MEDIUM: print
CONFERENCE/MEETING: 33rd Annual Meeting of the American Society of Nephrology and the 2000 Renal Week Toronto, Ontario, Canada October

10-16, 2000; 20001010
SPONSOR: American Society of Nephrology
ISSN: 1046-6673
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Other than the electrogenic Na+/K+-ATPase (BL or nutrient (N)
memb.) and H+/K+-ATPase (apical or secretory (S) memb.) pumps, the main
conductances (g's) in the oxynctic cells are: the Na-Cl (or NA-K-2Cl)
symport and the K+ g in the N memb. and the K+ and Cl- g's in the S memb.
Cuppoletti et al. have found recently (Ped. Pulm. Supp. 19, 1999)
that the COX inhibitor ibuprofen (IBU) increases the current in ClC-2 Cl
channels (S memb Cl- g). Present experiments were done using an in vitro
preparation of the frog gastric mucosa to study electrophysiological and
H+ secretion (H sec) effects of COX inhibitors. With addition of 5X10-4 M
IBU or 10-4 M meclofenamate (MEC) to the N soln, the short circuit (Isc)
decreased, in 20min, by 45 from 56 muA/1.3 cm2; the transepithelial
resistance (R) increased by 190 from 437 OMEGAXcm2; the transepithelial
potential (PD) decreased by 18 from 26 mV; and H+ secretion (H sec) was
not affected. In order to study the effect of IBU and MEC on the ionic
partial conductivity's (pg), the ion substitution method was used. While both
COX inhibitors decreased significantly the pg's of the Na-Cl symport and
K+ in the N memb. and the K+ pg in the S memb., the Cl- pg in the S memb
increased by 50% with IBU and was not affected by MEC. The increase of
the Cl- pg in the S memb by IBU in the in vitro gastric mucosa confirms
the findings on the Cl- channel by Cuppoletti et al. The disruptive
effects on the pg's, Isc, R, and PD by the two inhibitors, support the
well known deleterious effects that, particularly, inhibitors of
COX-1 have on the stomach and the kidney. The lack of effect on H sec
suggests that neither of the Na+ or H+ pumps were affected by these
inhibitors.

17/7/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16627057 BIOSIS NO.: 200200220568
Three hydrophobic amino acids in the factor VIII C2 domain mediate interactions with phospholipid and with factor IXa
AUTHOR: Gilbert Gary E (Reprint); Kaufman Randal J; Ahmed Ishtiaq (Reprint); Miao Hongzhi; Pipe Steven
AUTHOR ADDRESS: Medicine Depts, VA Boston Healthcare System, Brigham and Women's Hosp, Harvard Med. School, Boston, MA, USA**USA
JOURNAL: Blood 98 (11 Part 1): p705a November 16, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: 43rd Annual Meeting of the American Society of Hematology, Part 1 Orlando, Florida, USA December 07-11, 2001; 20011207
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Factor VIII (fVIII) binds to phospholipid (PL) membranes, to von Willebrand factor (vWf) and to factor IXa (Gilbert, Thromb. Haemost. 2001 Supp OC1659) via motifs localized to the C2 domain. We have shown that PL binding and vWf binding are mediated by two pairs of hydrophobic residues, each displayed at the tips of beta-hairpin turns. The homologous hydrophobic residues in the C2 domain of factor V also contribute to PL binding. We hypothesized that the solvent-exposed hydrophobic residues of the fVIII C2 domain make specific contacts with both PL and factor IXa rather than merely providing hydrophobic surface area. To test this hypothesis we have prepared 3 fVIII mutants in which amino acid(s) were changed to the homologous residues of factor V. Mutants were expressed in COS cells and protein purified by immunoaffinity chromatography. Mutants 1-M/F 2199/2200 W/W, 2-L/L 2251/2252 L/S, and 3-M/F/L 2199/2200/2252 W/W/S had specific activity in the range of 90-180% of wild type fVIII in both 1-stage and 2-stage commercial aPTT assays that contain a large excess of PL. In a PL-limiting Xase assay (sonicated vesicles of PS:PE:PC 4:20:76, 0.15 muM PL) the mutants had >95% (1), >95% (2), and 85% (3) reduction in specific

activity. Phospholipid titration indicated that maximum activity for the mutants occurred at concentrations of 800 (1), 800 (2), and 200 muM (3) vs. 4 muM for wild type fVIII. In a Xase assay with saturating PL, 1000 muM, the apparent affinity of factor IXa for the mutants was decreased approximately 4-fold for the 3 mutants and the maximum catalytic rate decreased by approximately 50 (1), 80 (2), and 50% (3). When the PS content was increased from 4% to 15% PS, all 3 mutants supported Xase activity within 60% of wild type fVIII although the apparent affinity for phospholipid was reduced 4-8 fold and for factor IXa was reduced 5-fold. Together these results indicate that the hydrophobic spikes composed of M/F2199/2200 and L/L2251/2252 mediate interactions with both phospholipid and factor IXa that are distinct from those of the homologous residues of factor V. Equal or increased activity of M/F/L 2199/2200/2252 W/W/S vs. either mutant in which a single hydrophobic pair was altered suggests that the two hydrophobic pairs may interact cooperatively in the presence of PL and factor IXa.

17/7/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15616718 BIOSIS NO.: 200000335031
Influence of pathogenicity islands and the minor leuX-encoded tRNA5Leu on the proteome pattern of the uropathogenic Escherichia coli strain 536
AUTHOR: Piechaczek Katharine; Dobrindt Ulrich; Schierhorn Angelika; Fischer Gunter S; Hecker Michael; Hacker Joerg (Reprint)
AUTHOR ADDRESS: Institut fuer Molekulare Infektionsbiologie, Roentgenring 11, D-97070, Wuerzburg, Germany**Germany
JOURNAL: IJMM International Journal of Medical Microbiology 290 (1): p 75-84 March, 2000 2000
MEDIUM: print
ISSN: 1438-4221
DOCUMENT TYPE: Article

RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The uropathogenic Escherichia coli strain 536 (O6:K15:H31) carries four distinct DNA regions in its chromosome, termed pathogenicity islands (PAIs I536 to IV536). Each of these PAIs encodes at least one virulence factor. All four PAIs are associated with tRNA genes. PAI I536 and PAI II536 can be spontaneously deleted from the chromosome by homologous recombination between flanking direct repeats. The deletion of PAI II536 results in the truncation of the associated gene leuX encoding the tRNA5Leu. This tRNA influences the expression of various virulence traits. In order to get a deeper insight into the role of PAI I536/II536 and of the tRNA5Leu for the protein expression, the protein expression patterns of Escherichia coli 536 and different derivatives were studied. Differences in the protein expression patterns of the wild-type strain Escherichia coli 536, its mutants 536-21 (PAI I536-, PAI II536-, leuX-), 536DELTA102 (PAI I536+, PAI II536+, leuX-) as well as of the strain 536R3 (PAI I536-, PAI II536-, leuX+) were analyzed by two-dimensional polyacrylamide gel electrophoresis and MALDI-TOF mass spectrometry. We identified about 39 different intracellular proteins whose expression is markedly altered in the different strain backgrounds. These differences can be linked either to the presence or absence of the PAI I536 and PAI II536 or to that of the tRNA gene leuX. The identities of 34 proteins have been determined by MALDI-TOF-MS. The identification of five proteins was not possible. The results suggest that proteome analysis is an efficient approach to study differences in global gene expression. The comparison of protein expression patterns of the uropathogenic E. coli strain 536 and different derivatives revealed that in this strain the expression of various proteins including those encoded by many housekeeping genes is affected by the presence of PAI I536 and Pai II536 or by that of the tRNA5Leu.

17/7/9 (Item 9 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15307143 BIOSIS NO.: 200000025456
Microglia only weakly present glioma antigen to cytotoxic T cells
AUTHOR: Fluegel Alexander; Labeur Marta S; Grasbon-Frodl Eva-Maria; Kreutzberg Georg W; Graeber Manuel B (Reprint)
AUTHOR ADDRESS: Department of Neuromorphology, Max-Planck-Institute of Neurobiology, Am Klopferspitz 18a, 82152, Martinsried, Germany**Germany
JOURNAL: International Journal of Developmental Neuroscience 17 (5-6): p
547-556 Aug.-Oct., 1999 1999
MEDIUM: print
ISSN: 0736-5748
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Microglia and brain macrophages represent a substantial fraction of the cells present in astrocytic gliomas. Yet, the functional role of microglia in these tumors has remained enigmatic. We have compared rat microglial cells and thymocytes with regard to their ability to present purified CNS proteins, MBP and S100beta, as well as C6 glioma cells to specific T lymphocytes. In addition, a new cytotoxicity assay based on fluorescence activated cell sorting of tumor cells carrying the green fluorescent protein was established. This assay was used to determine the influence of microglial population density and activational state on C6 glioma cell survival in vitro. Microglia were consistently found to present MBP and S100beta less efficiently than thymocytes and appeared to be unable to present C6 glioma cells to cytotoxic T lymphocytes. In addition, high concentrations of microglial cells attenuated the cytotoxic effects of these T cells on C6 glioma cells whereas thymocytes significantly supp orted their specific killing. It is suggested that defense functions of microglial cells against C6 glioma are severely compromised and that the observed deficiency in antigen presentation may play an important role for astrocytoma growth in vivo.

17/7/10 (Item 10 from file: 5)

14524127 BIOSIS NO.: 199800318374
Novel temperature-sensitive mutants of Escherichia coli that are unable to grow in the absence of wild-type tRNA6Leu
AUTHOR: Nakayashiki Toru; Inokuchi Hachiro (Reprint)
AUTHOR ADDRESS: Dep. Biophysics, Faculty Science, Kyoto Univ., Sakyo-ku, Kyogo 606-8502, Japan**Japan
JOURNAL: Journal of Bacteriology 180 (11): p2931-2935 June, 1998 1998
MEDIUM: print
ISSN: 0021-9193
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Escherichia coli has only a single copy of a gene for tRNA6Leu (Y. Komine et al., J. Mol. Biol. 212:579-598, 1990). The anticodon of this tRNA is CAA (the wobble position C is modified to O2-methylcytidine), and it recognizes the codon UUG. Since UUG is also recognized by tRNA4Leu, which has UAA (the wobble position U is modified to 5-carboxymethylaminomethyl-O2-methyluridine) as its anticodon, tRNA6Leu is not essential for protein synthesis. The BT63 strain has a mutation in the anticodon of tRNA6Leu with a change from CAA to CUA, which results in the amber suppressor activity of this strain (supP, Su+6). We isolated 18 temperature-sensitive (ts) mutants of the BT63 strain whose temperature sensitivity was complemented by introduction of the wild-type gene for tRNA6Leu. These tRNA6Leu-requiring mutants were classified into two groups. The 10 group I mutants had a mutation in the miaA gene, whose product is involved in a modification of tRNAs that stabilizes codon-anticodon interactions. Overexpression of the gene for tRNA4Leu restored the growth of group I mutants at 42degree C. Replacement of the CUG codon with UUG reduced the efficiency of translation in group I mutants. These results suggest that unmodified tRNA4Leu poorly recognizes the UUG codon at 42degree C and that the wild-type tRNA6Leu is required for translation in order to maintain cell viability. The mutations in the six group II mutants were complemented by introduction of the gidA gene, which may be involved

in cell division. The reduced efficiency of translation caused by replacement of the CUG codon with UUG was also observed in group II mutants. The mechanism of requirement for tRNA6Leu remains to be investigated.

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Set     Items   Description
S1        581   (ATTENUAT? OR AVIRULENT OR VACCIN?) AND SALMONELLA
AND (PAI
               OR LEUX OR PATHOGENICITY(W)ISLAND)
S2        174   RD S1  (unique items)
S3        139   S2 NOT PY>2006
S4          0   S3 AND (LEUX OR TRNA5LEU)
S5          1   S3 AND TRNA
S6          0   S3 AND SUPP
S7        462   (ATTENUAT? OR AVIRULENT OR VACCIN?) AND (TYPHI OR
DUBLIN OR
               TYPHIMURIUM) AND (PAI OR LEUX OR PATHOGENICITY(W)ISLAND)
S8        163   RD S7  (unique items)
S9          1   S8 AND (LEUX OR TRNA OR SUPP)
S10       113   E1-E5
S11        46   AU='COHEN, P. S.'
S12       159   S10 OR S11
S13         3   S12 AND (LEUX OR TRNA OR SUPP)
S14      3901   (LEUX OR TRNA5LEU OR SUPP)
S15      3349   RD S14  (unique items)
S16      2662   S15 NOT PY>2005
S17        87   S16 AND (DELET? OR MUTAT? OR VARIANT? OR MUTEIN OR
AVIRULE-
              NT OR ATTENUAT?)
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>>>Format 7 is not valid in file 143

 17/7/11     (Item 11 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
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14478602   BIOSIS NO.: 199800272849
The leuX-encoded tRNA5Leu but not the pathogenicity islands I
  and II influence the survival of the uropathogenic Escherichia coli
  strain 536 in CD-1 mouse bladder mucus in the stationary phase
AUTHOR: Dobrindt Ulrich; Cohen Paul S; Utley Maryjane; Muehldorfer
Inge;
  Hacker Joerg (Reprint)
AUTHOR ADDRESS: Inst. Mol. Infektionsbiol., Univ. Wuerzburg,
Roentgenring
  11, 97070 Wuerzburg, Germany**Germany
JOURNAL: FEMS Microbiology Letters  162 (1): p135-141 May 1, 1998 1998
MEDIUM: print
ISSN: 0378-1097
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
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LANGUAGE: English

ABSTRACT: The uropathogenic Escherichia coli strain 536 carries two pathogenicity islands, each of which is associated with either of the tRNA genes selC or leuX, respectively. Growth competition in CD-1 mouse mucus between the wild-type strain E. coli 536, its leuX mutant 536DELTA102 and its mutant 536R3, lacking both pathogenicity islands but expressing a functional tRNA5Leu, revealed a major impact of leuX on E. coli survival in bladder mucus. The impaired survival in CD-1 mouse mucus observed upon deletion of the leuX gene was abolished after complementation with the leuX gene. The survival of bacteria in bladder mucus was not influenced by the presence of pathogenicity islands I and II.

17/7/12 (Item 12 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14151982 BIOSIS NO.: 199799786042
The Pai-associated leuX specific tRNA-5-Leu affects type 1 fimbriation in pathogenic Escherichia coli by control of FimB recombinase expression
AUTHOR: Ritter Angelika; Gally David L; Olsen Peter B; Dobrindt Ulrich; Friedrich Arne; Klemm Per; Hacker Joerg (Reprint)
AUTHOR ADDRESS: Inst. Molekulare Infektionsbiologie, Roentgenring 11, D-97070 Wurzburg, Germany**Germany
JOURNAL: Molecular Microbiology 25 (5): p871-882 1997 1997
ISSN: 0950-382X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The uropathogenic Escherichia coli strain 536 (O6:K15: H31) carries two large chromosomal pathogenicity islands (Pals). Both Pais are flanked by tRNA genes. Spontaneous deletion of Pal II results in truncation of the leuX tRNA-5-Leu gene. This tRNA is required for the expression of type 1 fimbriae (Fim) and other virulence factors. Transcription of fimA, encoding the major type 1 fimbrial subunit is controlled by an invertable DNA switch. The inversion is catalysed by two recombinases, FimB and FimE. FimB is able to turn the switch on, FimE only off. The fimB gene of strain 536 contains five TTG codons recognized by tRNA-5-Leu fimE contains only two. It was proposed that turning on the

fim switch requires efficient translation of FimB, in turn requiring tRNA-5-Leu. Strains in which the TTG codons in fimB were replaced with CTG codons at the wild-type locus were able to produce type 1 fimbriae in the absence of leuX. fimB transcription was influenced by the presence of leuX, but only slightly affected by the presence or absence of leuX codons in fimB. FimB translation was significantly higher from codon-replaced fimB genes than that of wild type fimB genes in various strain backgrounds. The fim switch was shown to be switched off in leuX- derivatives of E. coli 536, but could be found in the on position when the codon-altered fimB gene was exchanged into the chromosome of these strains. From these data, it is apparent that tRNA-5-Leu is required for efficient translation of FimB, in turn, leading to type 1 fimbrial expression.

17/7/13 (Item 13 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12118001 BIOSIS NO.: 199497139286
Excision of large DNA regions termed pathogenicity islands from tRNA-specific loci in the chromosome of an Escherichia coli wild-type pathogen
AUTHOR: Blum Gabriele; Ott Manfred; Lischewski Axel; Ritter Angelika; Imrich Horst; Tschaepe Helmut; Hacker Joerg (Reprint)
AUTHOR ADDRESS: Inst. Mol. Infektionsbiol., Univ. Wuerzburg, Roentgenring 11, D-97070 Wuerzburg, Germany**Germany
JOURNAL: Infection and Immunity 62 (2): p606-614 1994 1994
ISSN: 0019-9567
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Uropathogenic Escherichia coli 536 (O6:K15:H31) carries two unstable DNA regions, which were shown to be responsible for virulence. These regions, on which the genes for hemolysin production (hly) and P-related fimbriae (prf) are located, are termed pathogenicity islands (PAI) I and II, and were mapped to positions 82 and 97, respectively, on the E. coli K-12 linkage map. Sequence analysis of the PAI region junction sites revealed sequences of the leuX and selC loci specific for leucine and selenocysteine tRNAs. The tRNA loci function as the targets for excision events. Northern (RNA) blot analysis revealed

that the sites of excision are transcriptionally active in the wild-type strain and that no tRNA-specific transcripts were found in the deletion mutant. The analysis of deletion mutants revealed that the excision of these regions is specific and involves direct repeats of 16 and 18 nucleotides, respectively, on both sides of the deletions. By using DNA long-range mapping techniques, the size of PAI 1, located at position 82, was calculated to be 70 kb, while PAI II, mapped at position 97, comprises 190 kb. The excision events described here reflect the dynamics of the E. coli chromosome.

17/7/14 (Item 14 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

10689364 BIOSIS NO.: 199191072255
TEMPERATURE SENSITIVITY CAUSED BY MISSENSE SUPPRESSOR SUPH AND AMBER SUPPRESSOR SUPF IN ESCHERICHIA-COLI
AUTHOR: THORBJARNARDOTTIR S (Reprint); BJORNSSON A; AMUNDADOTTIR L; EGGERTSSON G
AUTHOR ADDRESS: INST BIOL, UNIV ICELAND, 108 REYKJAVIK, ICELAND**ICELAND
JOURNAL: Journal of Bacteriology 173 (1): p412-416 1991
ISSN: 0021-9193
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

ABSTRACT: The temperature-sensitive missense suppressor supH and amber suppressor supP in Escherichia coli are mutations of the serU and leuX genes, respectively. The supH tRNA, tRNACAASER, is expected to recognize UUG codons, which are normally read by the tRNACAALeu and tRNAUAALeu, coded for bu the leuX gene and the leuZ gene, respectively. We show that supP and supH are incompatible and that strains carrying both supP and a restrictive rpsL allele are temperature sensitive. It is suggested that the temperature sensitivity of both supH and supP strains is caused by deficient reading of UUG codons by tRNAUAALeu.

17/7/15 (Item 15 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

10667128 BIOSIS NO.: 199191050019
IDENTIFICATION OF A DNA SEQUENCE RESPONSIBLE FOR BINDING OF THE 1 25 DIHYDROXYVITAMIN D-3 RECEPTOR AND 1 25 DIHYDROXYVITAMIN D-3 ENHANCEMENT

OF MOUSE SECRETED PHOSPHOPROTEIN 1 SPP-1 OR OSTEOPONTIN GENE EXPRESSION
AUTHOR: NODA M (Reprint); VOGEL R L; CRAIG A M; PRAHL J; DELUCA H F; DENHARDT D T
AUTHOR ADDRESS: DEP BONE BIOLOGY OSTEOPOROSIS RESEARCH, MERCK SHARP DOHME RESEARCH LABORATORIES, WEST POINT, PA 19486, USA**USA
JOURNAL: Proceedings of the National Academy of Sciences of the United States of America 87 (24): p9995-9999 1990
ISSN: 0027-8424
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

ABSTRACT: Secreted phosphoprotein 12 (Supp-1; osteopontin) is one of the abundant noncollagenous proteins in bone matrix and is produced by osteoblasts. We examined the promoter region of the mouse Spp-1 gene and identified a sequence responsible for 1,25-dihydroxyvitamin D3 enhancement of the Spp-1 gene expression. This 24-base-pair (bp) sequence (vitamin D response element) is located 761 bp upstream of the transcription start site and consists of two direct repeats of a unique 9-bp motif, AGGTTCACG. The vitamin D response element confers responsiveness of a heterologous promoter to 1,25-dihydroxyvitamin D3 in a position- and orientation-independent and copy-number-dependent manner. The basal level of expression of the receptor constructs containing this sequence and its response to 1,25-dihydroxyvitamin D3 were not affected by cotreatment with transforming growth factor β or the tumor promoter phorbol 12-myristate 13-acetate or by cotransfection with a JUN expression vector. The vitamin D response element forms DNA-protein complexes, as indicated by gel-retardation assays. The addition of a monoclonal antibody raised against the vitamin D receptor further retarded the mobility of the DNA-protein complex. Another antibody that recognizes the DNA binding region of the vitamin D receptor attenuated its binding to the sequence. These results indicate that this 24-bp sequence containing two 9-bp motifs binds to the vitamin D receptor and mediates the vitamin D3 enchancement of murine Spp-1 gene expression.

09715504 BIOSIS NO.: 198988030619
PCLODF13-ENCODED BACTERIOCIN RELEASE PROTEINS WITH SHORTENED CARBOXYL-TERMINAL SEGMENTS ARE LIPID MODIFIED AND PROCESSED AND FUNCTION IN RELEASE OF CLOACIN DF13 AND APPARENT HOST CELL LYSIS
AUTHOR: LUIRINK J (Reprint); CLARK D M; RAS J; VERSCHOOR E J; STEGEHUIS F; DE GRAAF F K; OUDEGA B
AUTHOR ADDRESS: DEP MOL MICROBIOL, BIOL LAB, VRIJE UNIV, DE BOELELAAN 1087, 1081 HV AMSTERDAM, NETHERLANDS**NETHERLANDS
JOURNAL: Journal of Bacteriology 171 (5): p2673-2679 1989
ISSN: 0021-9193
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

ABSTRACT: By oligonucleotide-directed mutagenesis, stop codon mutations were introduced at various sites inthe pCloDF13-derived bacteriocin release protein (BRP) structural gene. The expression, lipid modification (incorporation of [3H]palmitate), and processing (in the presence and absence of globomycin) of the various carboxyl-terminal shortened BRPs were analyzed by a special electorphoresis system and immunoblotting with an antiserum raised against a synthetic BRP peptide, and their functioning with respect to release of cloacin DF13, lethality, and apparent host cell lysis were studied in Sup-, supF, and supP strains of Escherichia coli. All mutant BRPs were stably expressed, lipid modified, and processed by signal peptidase II, albeit with different efficiencies. The BRP signal peptide appeared to be extremely stable and accumulated in induced cells. Full induction of the mutant BRPs, including the shortest containing only 4 amino acid resudues of the mature polypeptide, resulted in phospholipase A-dependent and Mg2+-suppressible apparent cell lysis. The extent of this lysis varied withthe mutant BRP used. Induction of all mutant BRPs also prevented colony formation, whech appeared to be phospholipase A independent. One shortened BRP, containing 20 amino acid residues of the mature polypeptide, was still able to bring about the release of cloacin DF13.
The results indicated that the 8-amino-acid carboxyl-terminal segment of

the BRP contains a stong antigenic determinant and that a small segment between amino acid residues 17 and 21, located int eh carboxyl-terminal half of the BRP, is important for release of cloacin DF13. Either the stable signal peptide or the acylated amino-terminal BRP fragments (or both) are involved in host cell lysis and lethality.

17/7/17 (Item 17 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

07611742 BIOSIS NO.: 198579030641
IDENTIFICATION OF TRANSFER RNA SUPPRESSORS IN ESCHERICHIA-COLI 4. AMBER SUPPRESSOR SU-PLUS-6 A DOUBLE MUTANT OF A NEW SPECIES OF LEUCINE TRANSFER RNA
AUTHOR: YOSHIMURA M (Reprint); INOKUCHI H; OZEKI H
AUTHOR ADDRESS: DEP BIOPHYS, FAC SCI, KYOTO UNIV, KYOTO 606, JPN**JAPAN
JOURNAL: Journal of Molecular Biology 177 (4): p627-644 1984
ISSN: 0022-2836
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

ABSTRACT: An E. coli DNA fragment containing an Su+6 amber suppressor gene (supP) was cloned into a λgtλCh vector by the shotgun method, selecting a Su+6 transducing phage λpSu+6. Through prophage integration followed by induction occurring at the transducing region of the λpSu+6 in Su- E. coli, a counterpart transducing phage carrying the wild-type allele (Su°6) was isolated (λpSu°6). The fingerprint of a tRNA encoded by λpSu°6 was identical to that of an unidentified tRNAE previously reported. The cloverleaf structure of this tRNA was determined by combining the results of tRNA analysis and DNA sequencing of the gene. Judging from the anticodon of 5'-CAA-3', Su°6 tRNA was identified as a new type of leucine isoacceptor in E. coli. Unlike other suppressors analyzed, Su+6 tRNA differed by 2 nucleotides from Su°6 tRNA; one at the anticodon (CAA to CUA) and the other at the junction of D- and anticodon-stem (A27 to G27). DNA sequence analysis revealed that a single

stretch of tRNA is flanked by the putative sequences of promoter and terminator. Thus, a single copy of the Su°6 tRNA gene constitutes a solitary tRNA transcription unit. Southern blotting showed only 1 copy of Su°6 tRNA gene/haploid genome of E. coli. Since this single gene can mutate to the Su+6 suppressor, the Su°6 leucine tRNA may be accounted as a dispensable species among the leucine isoacceptor tRNA species. Two possible open reading frames are found immediately following the Su°6 tRNA gene.

17/7/18 (Item 18 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

07328331 BIOSIS NO.: 198478063738
PURKINJE CELL ACTIVITY IN THE PRIMATE MACACA-MULATTA FLOCCULUS DURING OPTO
KINETIC STIMULATION SMOOTH PURSUIT EYE MOVEMENTS AND VESTIBULO OCULAR
REFLEX SUPPRESSION
AUTHOR: BUTTNER U (Reprint); WAESPE W
AUTHOR ADDRESS: DEP NEUROL, UNIV DUESSELDORF, MOORENSTR 5, D-4000 DUESSELDORF, W GER**WEST GERMANY
JOURNAL: Experimental Brain Research 55 (1): p97-104 1984
ISSN: 0014-4819
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

ABSTRACT: Purkinje cell (PC) activity in the flocculus of trained monkeys [M. mulatta] was recorded during: vestibular stimulation in darkness; suppression of the vestibulo-ocular reflex (VOR-supp) by fixation of a small light spot stationary with respect to the monkey; visual-vestibular conflict (i.e., the visual surround moves together with the monkey during vestibular stimulation), which leads to attenuation or suppression of vestibular nystagmus; smooth pursuit eye movements; optkinetic nystagmus (OKN); and suppression of nystagmus during optokinetic stimulation (OKN-supp) by fixation of a small light spot. Stimulus velocity corresponds then to image slip velocity.
Results were obtained from PC, which were activated with VOR-supp during rotation to the ipsilateral side. The same PC were also modulated during smooth pursuit and visual-vestibular conflict. No tonic modulation during constant velocity OKN occurred with slow-phase nystagmus velocities below 40-60 degrees/s. Tonic responses were only seen at

higher nystagmus velocities. Transient activity changes appeared at the beginning and end of optokinetic stimulation. PC were not modulated by image slip velocity during OKN-supp. In primates, the same population of floccular PC is involved in different mechanisms of visual-vestibular interaction. Smooth pursuit and certain components of OKN slow-phase velocity share the same neural pathway. The activity of these neurons can neither be related strictly to gaze, eye or image slip velocity; instead, their activity pattern can be best interpreted by assuming a modulation, which is complementary to that of central vestibular neurons of the vestibular nuclei, in the control of slow eye movements.

17/7/19 (Item 19 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

05397510 BIOSIS NO.: 197865058497
REGULATION OF BIOSYNTHESIS OF AMINOACYL TRANSFER RNA SYNTHETASES AND OF TRANSFER RNA IN ESCHERICHIA-COLI PART 3 BIOCHEMICAL CHARACTERIZATION OF REGULATORY MUTANTS AFFECTING LEUCYL TRANSFER RNA SYNTHETASE LEVELS
AUTHOR: LAROSSA R A (Reprint); MAO J-I; LOW K B; SOLL D
AUTHOR ADDRESS: DEP MOL BIOPHYS BIOCHEM, YALE UNIV, NEW HAVEN, CONN 06520, USA**USA
JOURNAL: Journal of Molecular Biology 117 (4): p1049-1060 1977
ISSN: 0022-2836
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

ABSTRACT: The consequences of 2 mutations, leuX and leuY, which affect the level of leucyl-tRNA synthetase on the amount of tRNA and other aminoacyl-tRNA syntheases and on the expression of the ilv and leu operons in E. coli were studied. Neither mutation appears to alter the cellular concentrations of other aminoacyl-tRNA syntheases or tRNA isoacceptor families. Leucyl-tRNA rather than leucyl-tRNA synthetase is the defective control element responsible for derepression of the leu and ilv operon in leuS31 strains. Steady-state levels of leucine, isoleucine,

valine, ppGpp, pppGpp and leucyl-tRNA, and the rate of protein synthesis were measured in leuS+, leuS-, leuS- leuX- and leuS- leuY- strains. The levels of magic spot compounds appear to correlate with the extent that protein synthesis is arrested rather than the concentration of limiting leucyl-tRNA.

17/7/20 (Item 20 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

04929674 BIOSIS NO.: 197662025813
MUTATIONAL PROPERTIES OF SUPP AMBER OCHRE SUPER SUPPRESSORS IN SACCHAROMYCES-CEREVISIAE
AUTHOR: GERLACH W L
JOURNAL: Molecular and General Genetics 144 (2): p213-215 1976
ISSN: 0026-8925
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: Unspecified

ABSTRACT: Mutational properties of the supP amber-ochre supersuppressor locus in S. cerevisiae are described. They are consistent with the proposition that the supP locus encodes a protein.

17/7/21 (Item 21 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

03392637 BIOSIS NO.: 197006079183
THE EFFECT OF LIVE ATTENUATED MEASLES VIRUS VACCINE ON THE CENTRAL NERVOUS SYSTEM
BOOK TITLE: BURDZY, KRYSTINA AND P. KALLOS (EDITED BY), INTERNATIONAL ARCHIVES OF ALLERGY AND APPLIED IMMUNOLOGY. VOL. 36. SUPP. PATHOGENESIS AND ETIOLOGY OF DEMYELINATING DISEASES. XI + 701P. ILLUS. S. KARGER: BASEL, SWITZERLAND AND NEW YORK, N.Y., U.S.A
AUTHOR: KATZ S L
p125-133 1969
DOCUMENT TYPE: Book
RECORD TYPE: Citation
LANGUAGE: Unspecified

17/7/22 (Item 22 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0001259504 BIOSIS NO.: 19624000007137
Lipase activity of the tubercle bacilli and atypic Mycobacteria [English
summ.]
ORIGINAL LANGUAGE TITLE: Activite lipasique des bacilles tubercu-leux et des Mycobacteries atypiques [English summ.]
AUTHOR: ANDRE JEW A; GERNEZ-RIEUX C; TACQUET A
AUTHOR ADDRESS: Inst. Pasteur Lille, France
JOURNAL: ANN INST PASTEUR [PARIS] 99 ((1)): p56-68 1960 1960
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: Unspecified

ABSTRACT: Quantitative studies on lipase activity (hydrolysis of Tweens)
and esterase activity (hydrolysis of tributyrin and triacetin) in various
mycobacteria is presented. A simple technic for the demonstration of
these activities is described. According to their lipase activity, the
mycobacteria can be divided into 2 main groups: mycobacteria possessing a
weak or no activity; mycobacteria possessing a strong activity. The
lipase activity is weak in all virulent and avirulent tubercle
bacilli (human, bovine and avian. The lipase activity of all the atypical
photochromogeneous mycobacteria is tenfold that of typical tubercle
bacilli. The lipase activity of mycobacteria belonging to other groups is
either weak (like that of typical t. b. or relatively strong (like that
of the photochromogeneous mycobacteria). Both "rapid growth" and
scotochromogeneous mycobacteria groups are heterogeneous: their lipase
activity is rather strong (M. phlei QMS 11), moderate (M. lacticola) or
weak (M. fortuitum R 322) according to the strains. Mycobacteria
possessing a strong lipase activity hydrolyze Tween 20 twice as rapidly
as the other Tweens. The findings are different with the simple
esterases: their activity is always rather strong whichever mycobacteria
is studied. ABSTRACT AUTHORS: Authors

17/7/23 (Item 1 from file: 24)
DIALOG(R)File 24:CSA Life Sciences Abstracts

0002744017 IP ACCESSION NO: 6426156
Multiple Insertional Events, Restricted by the Genetic Background, Have Led

to Acquisition of Pathogenicity Island II sub(J96)-Like Domains among Escherichia coli Strains of Different Clinical Origins

Bidet, Philippe; Bonacorsi, Stephane; Clermont, Olivier; De Montille,
Caroline; Brahimi, Naima; Bingen, Edouard
Laboratoire d'etudes de genetique bacterienne dans les infections de l'enfant (EA3105), Universite Denis Diderot-Paris 7, Service de Microbiologie, Hopital Robert Debre (AP-HP), 75019 Paris, France

Infection and Immunity, v 73, n 7, p 4081-4087, July 2005
PUBLICATION DATE: 2005

PUBLISHER: American Society for Microbiology, 1752 N Street N.W. Washington, DC 20036 USA, [URL:http://www.asm.org/]

DOCUMENT TYPE: Journal Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English
ISSN: 0019-9567
FILE SEGMENT: Bacteriology Abstracts (Microbiology B); Genetics Abstracts

ABSTRACT:
We investigated the dissemination of pathogenicity island (PAI) II sub(J96)-like elements (hra, hly, cnf1, and pap) among 455 Escherichia coli
isolates from children and adults with urinary tract infection (UTI), neonates with meningitis or colonized healthy neonates, and 74 reference
strains by means of PCR phylogenetic grouping, ribotyping, and PCR analysis
of virulence genes. Colocalization of these genes was documented by pulsed-field gel electrophoresis followed by Southern hybridization and
long-range PCR (LRPCR) between the hra and the papG alleles. Site-specific
insertion of the PAI was determined by LRPCR between hra and tRNA flanking
sequences. hra, hly, and cnf1 were found in 113 isolates and consistently
colocalized, constituting the backbone of PAI II sub(J96)-like domains. The
prevalence of PAI II sub(J96)-like domains was significantly higher among
UTI isolates than among neonatal meningitis and commensal isolates. These
domains were restricted to a few ribotypes of group B2. In contrast to the
consistent colocalization of hra, hly, and cnf1, the pap operon was varied:

12% of strains exhibited an allelic exchange of the papG class III allele
(papGIII) for the papG class II allele (papGII) (only UTI isolates), and
the pap operon was deleted in 23% of strains. No strains harbored
papGIII outside the PAI, which appears to be the only source of this
allele. PAI II sub(J96)-like domains were inserted in the vicinities of
three different tRNAs-pheU (54%), leuX (29%), and pheV
(15%)-depending on the genetic backgrounds and origins of the isolates.
Multiple insertional events restricted by the genetic background have thus
led to PAI II sub(J96) acquisition. Specific genetic backgrounds and
insertion sites may have played a role in additional recombination
processes for E. coli adaptation to different ecological niches.

17/7/24 (Item 2 from file: 24)
DIALOG(R)File 24:CSA Life Sciences Abstracts

0002464086 IP ACCESSION NO: 5594999
Transient IL-7/IL-7R Signaling Provides a Mechanism for Feedback Inhibition
of Immunoglobulin Heavy Chain Gene Rearrangements

Chowdhury, D; Sen, R
Rosenstiel Basic Medical Research Center and, Department of Biology,
Brandeis University, Waltham, MA 02454 USA, [mailto:sen@brandeis.edu]

Immunity, v 18, n 2, p 229-241, February 2003
PUBLICATION DATE: 2003

DOCUMENT TYPE: Journal Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English
ISSN: 1074-7613
FILE SEGMENT: Immunology Abstracts; Nucleic Acids Abstracts

ABSTRACT:
Production of immunoglobulin heavy chain (IgH) protein feeds back to
terminate further V sub(H) gene recombination, a phenomenon also referred
to as allelic exclusion. Here we provide evidence to supp the
proposition that allelic exclusion is the consequence of terminating
signals that activate V sub(H) genes recombination. For the largest V
sub(H)J558 family of genes, this occurs by attenuating IL-7/IL-7R
signals pre-B cells. Loss of these signals reverts the V sub(H) locus to a
chromatin state that is associated with hypoacetylated histones and
is less

accessible to nucleases. Furthermore, hyperacetylation and accessibility of
unrearranged V sub(H) genes can be restored in allelically excluded splenic
B cells by activating this pathway. Thus, transient signals mediate V sub(H) gene activation and inactivation during development.

17/7/25 (Item 3 from file: 24)
DIALOG(R)File 24:CSA Life Sciences Abstracts

0002431080 IP ACCESSION NO: 5524319
p53-dependent downregulation of metastasis-associated laminin receptor

Modugno, M; Tagliabue, E; Ardini, E; Berno, V; Galmozzi, E; De Bortoli, M; Castronovo, V; Menard, S
Molecular Targeting Unit, Department of Experimental Oncology, Instituto
Nazionale Tumori, Via Venezian 1, 20133 Milano, Italy,
[mailto:menard@institutotumori.mi.it]

Oncogene, v 21, n 49, p 7478-7487, October 24, 2002
PUBLICATION DATE: 2002

DOCUMENT TYPE: Journal Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English
ISSN: 0950-9232
DOI: 10.1038/sj.onc.1205957
FILE SEGMENT: Oncogenes & Growth Factors Abstracts

ABSTRACT:
Based on observations suggesting a role for the tumor suppressor protein
p53 in regulating expression of the 67-kDa laminin receptor precursor, 37LRP, we analysed the 37LRP promoter activity in a wild-type p53 (wt p53)
ovarian carcinoma cell line and in a cisplatin-resistant subline with mutated p53. We observed an increased promoter activity in wt p53 cells as compared to the mutated-p53 line when the first intron of the 37LRP gene was present in the reporter construct. Cotransfection experiments showed that the promoter is downregulated by both wt and mutated p53. Deletion analysis of the first intron localized an enhancer activity in the first 5' 214 bp that upregulates both 37LRP and
SV40 promoter activity and is repressed by both wt and mutant p53. Contransfection, mutagenesis and gel-shift experiments identified a functional AP-2 cis-acting element in this intron region that is repressed
by increased levels of both wt and mutated p53. Coimmunoprecipitation

studies revealed AP-2 in physical association in vivo with both wt and mutated p53, indicating for the first time that interaction of p53 with AP-2 is involved in the repression mechanism and in the regulation of
genes involved in cancer growth and progression.

17/7/26 (Item 4 from file: 24)
DIALOG(R)File 24:CSA Life Sciences Abstracts

0002335882 IP ACCESSION NO: 5400323
Efficient expression of the alpha -haemolysin determinant in the uropathogenic Escherichia coli strain 536 requires the leuX-encoded tRNA sub(5) super(Leu)

Dobrindt, U; Emoedy, L; Gentschev, I; Goebel, W; Hacker, J
Institut fuer Molekulare Infektionsbiologie, Universitaet Wuerzburg, Roentgenring 11, 97070 Wuerzburg, Germany

Molecular Genetics and Genomics, v 267, n 3, p 370-379, May 2002
PUBLICATION DATE: 2002

PUBLISHER: Springer-Verlag,
[URL:http://link.springer.de/link/service/journals/00438/bibs/2267003/22670370.htm]

DOCUMENT TYPE: Journal Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English
ISSN: 1617-4615
DOI: 10.1007/s00438-002-0668-3
FILE SEGMENT: Genetics Abstracts; Bacteriology Abstracts (Microbiology B)

ABSTRACT:
The uropathogenic Escherichia coli strain 536 (O6:K15:H31) carries two
alpha -haemolysin determinants which are located on different pathogenicity
islands (PAI I sub(536) and PAI II sub(536)). PAI II sub(536) is associated
with the tRNA gene leuX. The leuX-encoded tRNA sub(5) super(Leu) is required for the efficient expression of the hly determinants
in strain 536. HlyA levels were reduced and secretion of the protein was
delayed in the leuX-negative mutant strain 536 Delta 102. The lack of a functional tRNA sub(5) super(Leu) resulted in a decrease in hly transcript levels in comparison to the wild-type strain. Analysis of several genes whose products are involved in the regulation of hly

expression revealed that levels of RfaH and Hha, as well as the corresponding rfaH and hha transcripts, were higher in the leuX -negative background, whereas the expression of tolC and hns was not influenced by the leuX genotype. The analysis of hly transcript levels in hha deletion mutants of the E. coli strains 536 and 536 Delta 102 demonstrated that the increase in hha expression is partially
responsible for the reduction in hly transcript levels in the leuX -negative background. These results demonstrate that the tRNA sub(5) super(Leu) affects the expression of the alpha -haemolysin determinant at
different levels in a regulatory cascade, and imply that, in addition to
Hha, at least one further, as yet unidentified, regulatory factor must be
involved in the regulation of hly transcription in the uropathogenic E.
coli strain 536.

17/7/27 (Item 5 from file: 24)
DIALOG(R)File 24:CSA Life Sciences Abstracts

0002086151 IP ACCESSION NO: 4700763
Influence of pathogenicity islands and the minor leuX-encoded tRNA sub(5) super(Leu) on the proteome pattern of the uropathogenic Escherichia
coli strain 536

Piechaczek, K; Dobrindt, U; Schierhorn, A; Fischer, GS; Hecker, M; Hacker, J*
Institut fuer Molekulare Infektionsbiologie, Roentgenring 11, D-97070 Wuerzburg, Germany, [mailto:j.hacker@mail.uni-wuerzburg.de]

International Journal of Medical Microbiology, v 290, n 1, p 75-84, March
2000
PUBLICATION DATE: 2000

DOCUMENT TYPE: Journal Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English
ISSN: 1438-4221
FILE SEGMENT: Bacteriology Abstracts (Microbiology B)
ABSTRACT:
The uropathogenic Escherichia coli strain 536 (O6:K15:H31) carries four
distinct DNA regions in its chromosome, termed pathogenicity islands (PAIs
I sub(536) to IV sub(536)). Each of these PAIs encodes at least one

virulence factor. All four PAIs are associated with tRNA genes. PAI I sub(536) and PAI II sub(536) can be spontaneously deleted from the chromosome by homologous recombination between flanking direct repeats. The deletion of PAI II sub(536) results in the truncation of the associated gene leuX encoding the tRNA sub(5) super(Leu). This tRNA influences the expression of various virulence traits. In order to get a deeper insight into the role of PAI I sub(536)/II sub(536) and of the tRNA sub(5) super(Leu) for the protein expression, the protein expression patterns of Escherichia coli 536 and different derivatives were studied. Differences in the protein expression patterns of the wild-type strain Escherichia coli 536, its mutants 536-21 (PAI I sub(536) super(-), PAI II sub(536) super(-), leuX super(-)), 536 Delta 102 (PAI I sub(536) super(+), PAI II sub(536) super(+), leuX super(-)) as well as of the strain 536R3 (PAI I sub(536) super(-), PAI II sub(536) super(-), leuX super(+)) were analyzed by two-dimensional polyacrylamide gel electrophoresis and MALDI-TOF mass spectrometry. We identified about 39 different intracellular proteins whose expression is markedly altered in the different strain backgrounds. These differences can be linked either to the presence or absence of the PAI I sub(536) and PAI II sub(536) or to that of the tRNA gene leuX. The identities of 34 proteins have been determined by MALDI-TOF-MS. The identification of five proteins was not possible. The results suggest that proteome analysis is an efficient approach to study differences in global gene expression. The comparison of protein expression patterns of the uropathogenic E. coli strain 536 and different derivatives revealed that in this strain the expression of various proteins including those encoded by many housekeeping genes is affected by the presence of PAI I sub(536) and Pai II sub(536) or by that of the tRNA sub(5) super(Leu).

17/7/28 (Item 6 from file: 24)
DIALOG(R)File 24:CSA Life Sciences Abstracts

0001856956 IP ACCESSION NO: 4330946
Novel temperature-sensitive mutants of Escherichia coli that are unable to grow in the absence of wild-type tRNA sub(6) super(Leu)

Nakayashiki, T; Inokuchi, H
Department of Biophysics, Faculty of Science, Kyoto University,
Sakyo-ku,
Kyoto 606-8502, Japan, [mailto:00hachi@molbio.biophys.kyoto-u.ac.jp]

Journal of Bacteriology, v 180, n 11, p 2931-2935, June 1998
PUBLICATION DATE: 1998

DOCUMENT TYPE: Journal Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English
ISSN: 0021-9193
FILE SEGMENT: Genetics Abstracts; Bacteriology Abstracts
(Microbiology B);
Nucleic Acids Abstracts

ABSTRACT:
  Escherichia coli has only a single copy of a gene for tRNA sub(6)
super(Leu). The anticodon of this tRNA is CAA (the wobble position C
is
modified to O super(2)-methylcytidine), and it recognizes the codon
UUG.
Since UUG is also recognized by tRNA sub(4) super(Leu), which has UAA
(the
wobble position U is modified to 5-carboxymethylaminomethyl-O
super(2)-methyluridine) as its anticodon, tRNA sub(6) super(Leu) is
not
essential for protein synthesis. The BT63 strain has a mutation in
the anticodon of tRNA sub(6) super(Leu) with a change from CAA to CUA,
which results in the amber suppressor activity of this strain (supP,
Su super(+)6). We isolated 18 temperature-sensitive (ts) mutants of
the
BT63 strain whose temperature sensitivity was complemented by
introduction
of the wild-type gene for tRNA sub(6) super(Leu). These tRNA sub(6)
super(Leu)-requiring mutants were classified into two groups. The 10
group
I mutants had a mutation in the miaA gene, whose product is involved
in a modification of tRNAs that stabilizes codon-anticodon
interactions.
Overexpression of the gene for tRNA sub(4) super(Leu) restored the
growth
of group I mutants at 42 degree C. Replacement of the CUG codon with
UUG
reduced the efficiency of translation in group I mutants. These
results
suggest that unmodified tRNA sub(4) super(Leu) poorly recognizes the
UUG
codon at 42 degree C and that the wild-type tRNA sub(6) super(Leu) is
required for translation in order to maintain cell viability. The
mutations in the six group II mutants were complemented by

introduction of the gidA gene, which may be involved in cell division. The
reduced efficiency of translation caused by replacement of the CUG codon
with UUG was also observed in group II mutants. The mechanism of
requirement for tRNA sub(6) super(Leu) remains to be investigated.

17/7/29 (Item 7 from file: 24)
DIALOG(R)File 24:CSA Life Sciences Abstracts

0001833876 IP ACCESSION NO: 4293630
The leuX-encoded tRNA sub(5) super(Leu) but not the pathogenicity islands I and II influence the survival of the uropathogenic Escherichia
coli strain 536 in CD-1 mouse bladder mucus in the stationary phase

Dobrindt, U; Cohen, PS; Utley, M; Muhldorfer, I; Hacker, J
Institut fur Molekulare Infektionsbiologie, Universitat Wurzburg, Rontgenring 11, 97070 Wurzburg, Germany

FEMS Microbiology Letters, v 162, n 1, p 135-141, May 1, 1998
PUBLICATION DATE: 1998

PUBLISHER: Elsevier Science B.V.

DOCUMENT TYPE: Journal Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English
ISSN: 0378-1097
FILE SEGMENT: Bacteriology Abstracts (Microbiology B); Genetics Abstracts
ABSTRACT:
The uropathogenic Escherichia coli strain 536 carries two pathogenicity
islands, each of which is associated with either of the tRNA genes sel C or
leu X, respectively. Growth competition in CD-1 mouse mucus between the
wild-type strain E. coli 536, its leu X mutant 536 Delta 102 and its mutant
536R3, lacking both pathogenicity islands but expressing a functional tRNA
5 Leu, revealed a major impact of leu X on E. coli survival in bladder mucus. The impaired survival in CD-1 mouse mucus observed upon deletion of the leu X gene was abolished after complementation with the leu X gene. The survival of bacteria in bladder mucus was not influenced by the presence of pathogenicity islands I and II.

17/7/30 (Item 8 from file: 24)
DIALOG(R)File 24:CSA Life Sciences Abstracts

0001769275 IP ACCESSION NO: 4113846
The Pai-associated leuX specific tRNA sub(5) super(Leu) affects type 1 fimbriation in pathogenic Escherichia coli by control of FimB recombinase
expression

Ritter, A; Gally, DL; Olsen, PB; Dobrindt, U; Friedrich, A; Klemm, P;
Hacker, J*
Inst. fuer Molekulare Infektionsbiologie, Roentgenring 11, D-97070 Wuerzburg, FRG

Molecular Microbiology, v 25, n 5, p 871-882, September 1997
PUBLICATION DATE: 1997

DOCUMENT TYPE: Journal Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English
ISSN: 0950-382X
FILE SEGMENT: Bacteriology Abstracts (Microbiology B); Nucleic Acids Abstracts
ABSTRACT:
The uropathogenic Escherichia coli strain 536 (O6:K15:H31) carries two large chromosomal pathogenicity islands (Pais). Both Pais are flanked by tRNA genes. Spontaneous deletion of Pai II results in truncation of the leuX tRNA sub(5) super(Leu) gene. This tRNA is required for the expression of type 1 fimbriae (Fim) and other virulence factors. Transcription of fimA, encoding the major type 1 fimbrial subunit is controlled by an invertable DNA switch. The inversion is catalysed by two recombinases, FimB and FimE. FimB is able to turn the switch on, FimE only off. The fimB gene of strain 536 contains five TTG codons recognized by tRNA sub(5) super(Leu), fimE contains only two. It was proposed that turning on the fim switch requires efficient translation of FimB, in turn requiring tRNA sub(5) super(Leu). Strains in which the TTG codons in fimB were replaced with CTG codons at the wild-type locus were able to produce type 1 fimbriae in the absence of leuX. fimB transcription was influenced by the presence of leuX, but only slightly affected by the presence or absence of leuX codons in fimB. FimB translation was significantly higher from codon-replaced fimB genes than that of wild-type

fimB genes in various strain backgrounds. The fim switch was shown to be
switched off in leuX super(-) derivatives of E. coli 536, but could be found in the on position when the codon-altered fimB gene was exchanged
into the chromosome of these strains. From these data, it is apparent that
tRNA sub(5) super(Leu) is required for efficient translation of FimB, in
turn, leading to type 1 fimbrial expression.

17/7/31 (Item 9 from file: 24)
DIALOG(R)File 24:CSA Life Sciences Abstracts

0001277381 IP ACCESSION NO: 3003249
Temperature sensitivity caused by missense suppressor supH and amber suppressor supP in Escherichia coli .

Thorbjarnardottir, S; Bjoernsson, A; Amundadottir, L; Eggertsson, G
Inst. Biol., University Iceland, 108 Reykjavik, Iceland

Journal of Bacteriology, v 173, n 1, p 412-416, 1991
ADDL. SOURCE INFO: Journal of Bacteriology [J. BACTERIOL.], volume 173, number
1, pp. 412-416, 1991
PUBLICATION DATE: 1991

DOCUMENT TYPE: Journal Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English
ISSN: 0021-9193
FILE SEGMENT: Bacteriology Abstracts (Microbiology B)

ABSTRACT:
The temperature-sensitive missense suppressor supH and amber suppressor
supP in Escherichia coli are mutations of the serU and
leuX genes, respectively. The supH tRNA, tRNA sub(C) sub(A) sub(A) super(S)er)is expected to recognize UUG codons, which are normally read by
tRNA sub(C) sub(A) sub(A) super(L)eua)nd tRNA sub(U) sub(A) sub(A) super(L)eu)coded for by the leuX gene and the leuZ gene, respectively. We show that supP and supH are incompatible and that strains carrying both supP and a restrictive rpsL allele are temperature sensitive. It is suggested that the temperature sensitivity of
both supH and supP strains is caused by deficient reading of UUG codons by tRNA sub(U) sub(A) sub(A) super(L)eu)

17/7/32 (Item 10 from file: 24)
DIALOG(R)File 24:CSA Life Sciences Abstracts

0000256208 IP ACCESSION NO: 691386
Anticodon shift in tRNA: A novel mechanism in missense and nonsense suppression.

Murgoal, EJ; Prather, NE; Mims, BH; Pagel, FT; Hijazi, KA
Dep. Mol. Biol., University Texas M.D. Anderson Hosp. and Tumor Inst., Houston,
TX 77030, USA

Proceedings of the National Academy of Sciences, USA, v 80, n 16, p 4936-4939, 1983
ADDL. SOURCE INFO: Proceedings of the National Academy of Sciences, USA
[PROC. NATL. ACAD. SCI. USA.], vol. 80, no. 16, pp. 4936-4939, 1983
PUBLICATION DATE: 1983

CONFERENCE:
Gorini Memorial Symposium on Procaryotic Gene Expression, Lloyd Harbor, NY
(USA), 27 Jun-1 Jul 1982

DOCUMENT TYPE: Journal Article; Conference
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English
ISSN: 0027-8424
FILE SEGMENT: Nucleic Acids Abstracts; Genetics Abstracts; Bacteriology
Abstracts (Microbiology B)

ABSTRACT:
In a previous publication, an unusual UGG-reading missense suppressor
caused by insertion of an extra adenylate residue in the anticodon l oop of
an Escherichia coli glycine tRNA was described. In this st udy, the authors
provide in vito evidence that the additional nucleot ide causes an
"anticodon shift" by one nucleotide in the 3' direction and that the "new"
anticodon can explain the unanticipated coding pr operties of the
suppressor. They converted the UGG suppressor to supp ressors that
read codons related to UGG by a single base change. As d etermined on the
basis of their in vivo coding specificities, the new mutant tRNAs do not
continue to utilize the original anticodon tripl et for decoding.

Further-more, the failure of the UGG suppressor to c orrect frameshift mutations throughout each of three genes of the trp operon suggests that the addition of a nucleotide to the anticod on loop of a tRNA does not
necessarily result in out-of-frame decodin g by the tRNA. Therefore, a "frameshift" mutation in a tRNA has princ ipally changed the triplet codon recognition properties of the molecules.

17/7/33 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

07922877 Genuine Article#: 223XY Number of References: 6
Title: Apraxia of opening of the eyelids - Diagnostic and therapeutic problems
Author(s): Fiedler J; Kerndlova E
Corporate Source: NEUROL KLIN FN,/PLZEN//CZECH REPUBLIC/
Journal: CESKA A SLOVENSKA NEUROLOGIE A NEUROCHIRURGIE, 1999, V62, N4, P 226-228
ISSN: 1210-7859 Publication date: 19990000
Publisher: CZECH MEDICAL SOCIETY, SOKOLSKA 31, PRAGUE 2 120 26, CZECH REPUBLIC
Language: Czech Document Type: ARTICLE
Abstract: The authors discuss the differential diagnosis and treatment of so called atypical blepharospasm (apraxia of opening of the eyelids) as one of the not infrequent possible failures of treatment of blepharospasm. Atypical blepharospasm (AB) differs from idiopathic blepharospasm in particular by the temporary inability to start opening of the eyelids and the absence of an obvious spasm of the m. orbicularis oculi which is typical for idiopathic blepharospasm. In the investigated group the authors used botulotoxin A injections for 5 years in a total of 17 patients with blepharospasm, incl. 5 with the atypical variety, The injections were directed to the place of pretarsal portion of m, orbicularis oculi. The injections were strictly medial and lateral 2 on each upper eyelid up to its border - i.e. to the site where the eyelashes originate. The therapeutic effect was very satisfactory in the whole group of patients and when the mentioned procedure was respected the results in the atypical variant did not differ from those in classical idiopathic blepharospasm. In the authors' opinion the pathophysiological basis of the lesion in AB is a

disorder of the normal reciprocal alternation of activity of the m.
orbicularis oculi and the m. levator palpebrae supp,, but only in its pretarsal portion.

17/7/34 (Item 2 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

03078419 Genuine Article#: NE053 Number of References: 60
Title: FACTOR FOR INVERSION STIMULATION-DEPENDENT GROWTH-RATE REGULATION OF
INDIVIDUAL TRANSFER-RNA SPECIES IN ESCHERICHIA-COLI
Author(s): NILSSON L; EMILSSON V
Corporate Source: UPPSALA UNIV,CTR BIOMED,DEPT MOLEC BIOL/S-75124UPPSALA//SWEDEN/
Journal: JOURNAL OF BIOLOGICAL CHEMISTRY, 1994, V269, N13 (APR 1), P 9460-9465
ISSN: 0021-9258
Language: ENGLISH Document Type: ARTICLE
Abstract: We have studied the involvement of the factor for inversion stimulation (FIS) in the growth rate-dependent expression of the arginine, leucine, and methionine acceptor tRNA species. The concentration of individual tRNA species relative to 16 S rRNA was determined by blot hybridization using RNA preparations from bacteria with the fis gene deleted and from isogenic wild type bacteria. The RNA preparations were obtained from bacteria growing under steady state conditions in different media.

The levels of tRNA1Leu, tRNA2Arg, tRNA4Arg, and tRNA5Arg decreased in the fis bacteria, relative to the wild type. The difference in levels increased with increasing growth rate. Surprisingly, tRNA3Leu, tRNA(f)Met, and tRNA(e)Met showed the opposite response, with an increase of the tRNA/16 S ratio in the fis bacteria. The tRNA2Leu, tRNA4Leu, tRNA5Leu, and tRNA3Arg had unaffected tRNA/16 S ratios in fis cells. We conclude that FIS, directly or indirectly, is involved in growth rate regulation of some tRNA species and that it affects the composition of the cellular tRNA pool.

17/7/35 (Item 1 from file: 45)
DIALOG(R)File 45:EMCare

00406137 EMCare No: 127673855

Nociceptive and morphine antinociceptive sensitivity of 129 and C57BL/6
inbred mouse strains: Implications for transgenic knock-out studies

Mogil J.S.; Wilson S.G.
J.S. Mogil, Department of Psychology, University Illinois at Urbana-Champaign,
603 E Daniel Street, Champaign, IL 61820 United States

European Journal of Pain (EUR. J. PAIN) (United Kingdom) 1997, 1/4
(293-297)
CODEN: EJPAF ISSN: 1090-3801
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 15
RECORD TYPE: Abstract

Gene-targeting studies of pain, using transgenic 'knock-out' mice possessing null mutations of pain-relevant genes, are becoming increasingly common. This approach is a potentially powerful tool for the molecular dissection of complex traits such as pain modulation, but is subject to several theoretical drawbacks. One problem arises from the fact that the genetic background of knock-out mice is virtually always a mixture of alleles from two different strains; commonly 129 and C57BL/6. A more general caveat to knock-out findings derives from the demonstration that null mutations interact with genetic background to produce phenotypic changes. The present study investigated basal nociceptive sensitivity (on the 49degreesC tail-immersion/withdrawal test) and sensitivity to morphine antinociception in 129 and C57BL/6 mice (129/J, 129/Sv - +SUPp +SUPTyr-c+SUPMgf-SIJ, and C57BL/6J substrains). C57BL/6 mice displayed almost two-fold greater initial sensitivity to thermal stimulation than 129 mice, and three-fold reduced sensitivity to morphine inhibition of that noxious stimulus. These findings suggest that gene targeting studies of pain are particularly subject to the aforementioned concerns, and that C57BL/6 mice represent a suboptimal background strain for such efforts.

04201614 INSIDE CONFERENCE ITEM ID: CN044083759
Human gene mutation in pathology and evolution
Cooper, D. N.
CONFERENCE: Society for the Study of Inborn Errors of Metabolism-Annual
symposium; 39th
JOURNAL OF INHERITED METABOLIC DISEASE, 2002; VOL 25; NO 3 P: 157-182
Kluwer Academic, 2001
ISSN: 0141-8955
LANGUAGE: English DOCUMENT TYPE: Conference Papers and abstracts
CONFERENCE SPONSOR: Society for the Study of Inborn Errors of Metabolism
CONFERENCE LOCATION: Prague 2001; Sep (200109)
NOTE:
See also same s/m vol 24 supp 1 2001 for abstracts

17/7/37 (Item 2 from file: 65)
DIALOG(R)File 65:Inside Conferences

03946443 INSIDE CONFERENCE ITEM ID: CN041446808
Pathogenic Implication of Altered Tau Properties Caused by FTDP-17 Mutations
Nacharaju, P.; Yen, S.; DeTure, M.; Easson, C.; Hutton, M.; Yen, S.-H.
CONFERENCE: Alzheimer's disease and related disorders-International conference; 7th
P: 621-630
Chichester, New York, Wiley, 2001
ISBN: 0471521760
LANGUAGE: English DOCUMENT TYPE: Conference Selected papers
CONFERENCE EDITOR(S): Iqbal, K.; Sisodia, S. S.; Winblad, B.
CONFERENCE LOCATION: Washington, DC 2000; Jul (200007)
NOTE:
Held as part of the World Alzheimer's congress; See also shelfmark
6081.311 vol 21 supp 1 2000 for abstracts

17/7/38 (Item 3 from file: 65)
DIALOG(R)File 65:Inside Conferences

03946433 INSIDE CONFERENCE ITEM ID: CN041446705
Familial Alzheimer's Disease-linked Mutant Presenilins Attenuate Capacitative Calcium Entry
Cheng, I.; Yoo, A. S.; Tanzi, R. E.; Kim, T.-W.
CONFERENCE: Alzheimer's disease and related disorders-International conference; 7th
P: 515-520

Chichester, New York, Wiley, 2001
ISBN: 0471521760
LANGUAGE: English DOCUMENT TYPE: Conference Selected papers
CONFERENCE EDITOR(S): Iqbal, K.; Sisodia, S. S.; Winblad, B.
CONFERENCE LOCATION: Washington, DC 2000; Jul (200007)
NOTE:
Held as part of the World Alzheimer's congress; See also shelfmark
6081.311 vol 21 supp 1 2000 for abstracts

17/7/39 (Item 4 from file: 65)
DIALOG(R)File 65:Inside Conferences

03946429 INSIDE CONFERENCE ITEM ID: CN041446663
Intracellular and Secreted Abeta SUB 4 SUB 2 SUB / SUB 4 SUB 0 Ratios Are
Differently Influenced by APP Mutations
Grimm, H. S.; Lichtenthaler, S. F.; Beyreuther, K.; Hartmann, T.
CONFERENCE: Alzheimer's disease and related disorders-International conference; 7th
P: 479-486
Chichester, New York, Wiley, 2001
ISBN: 0471521760
LANGUAGE: English DOCUMENT TYPE: Conference Selected papers
CONFERENCE EDITOR(S): Iqbal, K.; Sisodia, S. S.; Winblad, B.
CONFERENCE LOCATION: Washington, DC 2000; Jul (200007)
NOTE:
Held as part of the World Alzheimer's congress; See also shelfmark
6081.311 vol 21 supp 1 2000 for abstracts

17/7/40 (Item 5 from file: 65)
DIALOG(R)File 65:Inside Conferences

03946428 INSIDE CONFERENCE ITEM ID: CN041446651
The Amyloid Precursor Protein V717I Mutation Increases Susceptibility to Cell Death in a Cholesterol-dependent Manner
Puglielli, L.; Ingano, L. A. M.; Tanzi, R. E.; Kovacs, D. M.
CONFERENCE: Alzheimer's disease and related disorders-International conference; 7th
P: 469-478
Chichester, New York, Wiley, 2001
ISBN: 0471521760
LANGUAGE: English DOCUMENT TYPE: Conference Selected papers
CONFERENCE EDITOR(S): Iqbal, K.; Sisodia, S. S.; Winblad, B.
CONFERENCE LOCATION: Washington, DC 2000; Jul (200007)
NOTE:
Held as part of the World Alzheimer's congress; See also shelfmark

6081.311 vol 21 supp 1 2000 for abstracts

17/7/41 (Item 6 from file: 65)
DIALOG(R)File 65:Inside Conferences

03946392 INSIDE CONFERENCE ITEM ID: CN041446298
Familial Alzheimer's Disease with Spastic Paraparesis Associated with a
Mutation at Codon 261 of the Presenilin 1 Gene
Farlow, M. R.; Murrell, J. R.; Unverzagt, F. W.; Phillips, M.; Takao, M.
; Hulette, C.; Ghetti, B.
CONFERENCE: Alzheimer's disease and related disorders-International conference; 7th
P: 53-60
Chichester, New York, Wiley, 2001
ISBN: 0471521760
LANGUAGE: English DOCUMENT TYPE: Conference Selected papers
CONFERENCE EDITOR(S): Iqbal, K.; Sisodia, S. S.; Winblad, B.
CONFERENCE LOCATION: Washington, DC 2000; Jul (200007)
NOTE:
Held as part of the World Alzheimer's congress; See also shelfmark
6081.311 vol 21 supp 1 2000 for abstracts

17/7/42 (Item 7 from file: 65)
DIALOG(R)File 65:Inside Conferences

03787841 INSIDE CONFERENCE ITEM ID: CN039808687
TAU GENE MUTATIONS AND TAU PATHOLOGY IN FRONTOTEMPORAL DEMENTIA AND PARKINSONISM LINKED TO CHROMOSOME 17
Spillantini, M. G.; Goedert, M.
CONFERENCE: Swiss Society of Neuropathology-International winter meeting;
18th
ADVANCES IN EXPERIMENTAL MEDICINE AND BIOLOGY, 2001; VOL 487 P: 21-38
New York, Kluwer Academic/Plenum Publishers, 2001
ISSN: 0065-2598 ISBN: 0306465582
LANGUAGE: English DOCUMENT TYPE: Conference Papers
CONFERENCE EDITOR(S): Tolnay, M.; Probst, A.
CONFERENCE SPONSOR: Swiss Society of Neuropathology
CONFERENCE LOCATION: St. Moritz, Switzerland 2000; Mar (200003)
NOTE:
See also 0806.25536 vol 3 supp 1 2000 for abstracts

17/7/43 (Item 1 from file: 71)

03016787 2005175815

Multiple insertional events, restricted by the genetic background, have led to acquisition of pathogenicity island IISUBJ96-like domains among Escherichia coli strains of different clinical origins

Bidet P.; Bonacorsi S.; Clermont O.; De Montille C.; Brahimi N.; Bingen E.
ADDRESS: E. Bingen, Service de Microbiologie, Hopital Robert Debre, 48 Bd. Serurier, 75395 Paris Cedex 19, France
EMAIL: edouard.bingen@rdb.ap-hop-paris.fr

Journal: Infection and Immunity, 73/7 (4081-4087), 2005, United States
CODEN: INFIB
ISSN: 0019-9567
DOCUMENT TYPE: Article
LANGUAGES: English SUMMARY LANGUAGES: English
NO. OF REFERENCES: 41

We investigated the dissemination of pathogenicity island (PAI) II SUBJ96-like elements (hra, hly, cnf1, and pap) among 455 Escherichia coli isolates from children and adults with urinary tract infection (UTI), neonates with meningitis or colonized healthy neonates, and 74 reference strains by means of PCR phylogenetic grouping, ribotyping, and PCR analysis of virulence genes. Colocalization of these genes was documented by pulsed-field gel electrophoresis followed by Southern hybridization and long-range PCR (LRPCR) between the hra and the papG alleles. Site-specific insertion of the PAI was determined by LRPCR between hra and tRNA flanking sequences, hra, hly, and cnf1 were found in 113 isolates and consistently colocalized, constituting the backbone of PAI IISUBJ96-like domains. The prevalence of PAI II SUBJ96-like domains was significantly higher among UTI isolates than among neonatal meningitis and commensal isolates. These domains were restricted to a few ribotypes of group B2. In contrast to the consistent colocalization of hra, hly, and cnf1, the pap operon was varied: 12% of strains exhibited an allelic exchange of the papG class III allele (papGIII) for thepapG class II allele (papGII) (only UTI isolates), and the pap operon was deleted in 23% of strains. No strains harbored papGIII

outside the PAI, which appears to be the only source of this allele. PAI
IISUBJ96-like domains were inserted in the vicinities of three different
tRNAs-pheU (54%), leuX (29%), and pheV (15%)-depending on the genetic backgrounds and origins of the isolates. Multiple insertional events restricted by the genetic background have thus led to PAI IISUBJ96 acquisition. Specific genetic backgrounds and insertion sites may have played a role in additional recombination processes for E. coli adaptation
to different ecological niches.

17/7/44 (Item 2 from file: 71)
DIALOG(R)File 71:ELSEVIER BIOBASE

02941116 2005096817
Binding mode and transcriptional activation potential of high affinity ligands for the CBP KIX domain
Volkman H.M.; Rutledge S.E.; Schepartz A.
ADDRESS: A. Schepartz, Department of Chemistry, Yale University, New Haven,
CT 06520-8107, United States
EMAIL: Alanna.Schepartz@yale.edu
Journal: Journal of the American Chemical Society, 127/13 (4649-4658), 2005
, United States
PUBLICATION DATE: April 6, 2005
CODEN: JACSA
ISSN: 0002-7863
DOCUMENT TYPE: Article
LANGUAGES: English SUMMARY LANGUAGES: English
NO. OF REFERENCES: 37

We recently described a pair of ligands, PPKID4SUPP (4SUPP) and PPKID6SUPU
(6SUPU), which present the alpha-helical functional epitope found on helix
B of the CREB KID activation domain (KID SUPP) on a pancreatic fold protein scaffold 4SUPP and 6 SUPU bind the natural target of KIDSUPP, the
KIX domain of the coactivator CBP, with equilibrium dissociation constants
between 515 nM and 1.5 muM and compete effectively with KIDSUPP for binding
to CBP KIX (KIX).SUP1 Here we present a detailed investigation of the binding mode, orientation, and transcriptional activation potential of 4SUPP and 6SUPU. Equilibrium binding experiments using a panel of well-characterized KIX variants support a model in which 4SUPP binds

KIX in a manner that closely resembles that of KIDSUPP but 6SUPU binds an
overlapping, yet distinct region of the protein. Equilibrium binding experiments using a judiciously chosen panel of 4SUPP variants containing alanine or sarcosine substitutions along the putative alpha- or
PPII helix of 4SUPP support a model in which 4SUPP folds into a pancreatic
fold structure upon binding to KIX. Transcriptional activation assays performed in HEK293 cells using GAL4 DNA-binding domain fusion proteins
indicate that 4SUPP functions as a potent activator of p300/CBP-dependent
transcription. Notably, 6SUPU is a less potent transcriptional activator in
this context than 4SUPP despite the similarity of their affinities for CBP
KIX. This final result suggests that thermodynamic affinity is an important, although not exclusive, criterion controlling the level of KIX-dependent transcriptional activation. (c) 2005 American Chemical Society.

17/7/45 (Item 3 from file: 71)
DIALOG(R)File 71:ELSEVIER BIOBASE

02529677 2003317885
The Amino Acid Sequence SUP442GDASESUP446 in Na/K-ATPase Is an Important
Motif in Forming the High and Low Affinity ATP Binding Pockets
Imagawa T.; Kaya S.; Taniguchi K.
ADDRESS: T. Imagawa, Division of Chemistry, Graduate School of Science,
Hokkaido University, Kita-Ku, Kita 10 Nishi 8, Sapporo 060-0810,
Japan
EMAIL: toshi@sci.hokudai.ac.jp
Journal: Journal of Biological Chemistry, 278/50 (50283-50292), 2003,
United States
PUBLICATION DATE: December 12, 2003
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ISSN: 0021-9258
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LANGUAGES: English SUMMARY LANGUAGES: English
NO. OF REFERENCES: 54

A highly conserved amino acid sequence SUP442GDASESUP446 in the ATP binding
pocket of rat Na/K-ATPase was mutated, and the resulting proteins, G442A, G442P, D443A, S445A, and E446A, were expressed in HeLa cells to investigate the effect of individual ligands on Na/K-ATPase. The apparent

KSUBm for the high and low affinity ATP effects was estimated by ATP concentration dependence for the formation of the Na-dependent phosphoenzyme (KSUBmSUPh) and Na/K-ATPase activity (KSUBm SUP1). The apparent KSUBm for p-nitrophenylphosphate (pNPP) for K-dependent-pNPPase
(KSUBmSUPP) and its inhibition by ATP (KSUBi,0.5) and the apparent KSUBm
MgSUP2+, Na SUP+, KSUP+, and vanadate in Na/K-ATPase were also estimated.
For all the mutants, the value for ATP was (similar)2-10-fold larger than
that of the wild type. While the turnover number for Na/K-ATPase activity
were unaffected or reduced by 20(similar)50% in mutants G442(A/P) and D443A. Although both affinities for ATP effects were reduced as a result of
the mutations, the ratio, KSUBmSUP1/KSUBmSUPh, for each mutant was 1.3(similar)3.7, indicating that these mutations had a greater impact on the low affinity ATP effect than on the high affinity effect. Each KSUBm
SUPP value with the turnover number suggests that these
mutations favor the binding of pNPP over that of ATP. These data and others indicate that the sequence SUP442GDASESUP446 in the ATP binding pocket is an important motif that it is involved in both the high and low
affinity ATP effects rather than in free MgSUP2+, NaSUP+, and KSUP+ effects.

17/7/46 (Item 4 from file: 71)
DIALOG(R)File 71:ELSEVIER BIOBASE

02432610 2003216537
Mutations in the nomad retroelement are modifiers of position-effect variegation in Drosophila melanogaster
Whalen J.H.; O'Grady M.; Grigliatti T.A.
ADDRESS: J.H. Whalen, Department of Biological Sciences, Stanford University, Gilbert Laboratories 109, Stanford, CA 94305-5020,
United States
EMAIL: jhwhalen@stanford.edu
Journal: Chromosome Research, 11/6 (573-583), 2003, Netherlands
CODEN: CRRSE
ISSN: 0967-3849
DOCUMENT TYPE: Article
LANGUAGES: English SUMMARY LANGUAGES: English
NO. OF REFERENCES: 33

The E(var)63ASUPP mutation of Drosophila melanogaster was isolated in a genetic screen for P-element induced enhancers of wSUPm4 variegation.

Remobilization of the P-element in E(var)63ASUPP resulted in a loss of its ability to enhance position-effect variegation (PEV) of w SUPm4, indicating that the P-element in this mutant resulted in the E(var) phenotype. An allele of E(var)63ASUPP, Su(var)63ASUPLTR was isolated following mobilization of the P-element. Su(var)63ASUPLTR was demonstrated to suppress PEV associated with the variegating rearrangements wSUPm4 and bwSUPDe2. The P-element insert in E(var)63A SUPP was located in the cytogenetic region 63A by in-situ hybridization and was shown to be inserted into the 3primeLTR of a copy of the nomad retroelement. Two additional P-element containing lines were identified that also contained P-inserts into copies of the nomad element and were Su(var)s. The level of nomad transcription in the E(var)63ASUPP and Su(var)63ASUPLTR mutations was shown to correlate with their effect on PEV, suggesting that the nomad element may be directly involved in the regulation of chromatin structure. Several models to explain the effect of mutations in the nomad element on PEV and retroelement expression are presented.

17/7/47 (Item 5 from file: 71)
DIALOG(R)File 71:ELSEVIER BIOBASE

02044678 2002125480
Efficient expression of the alpha-haemolysin determinant in the uropathogenic Escherichia coli strain 536 requires the leuX-encoded tRNASUB5SUPLeu
Dobrindt U.; Emody L.; Gentschev I.; Goebel W.; Hacker J.
ADDRESS: J. Hacker, Inst. fur Molek. Infektionsbiologie, Universitat Wurzburg, Rontgenring 11, 97070 Wurzburg, Germany
EMAIL: j.hacker@mail.uni-wuerzburg.de
Journal: Molecular Genetics and Genomics, 267/3 (370-379), 2002, Germany
CODEN: MGGOA
ISSN: 1617-4615
DOCUMENT TYPE: Article
LANGUAGES: English SUMMARY LANGUAGES: English
NO. OF REFERENCES: 43

The uropathogenic Escherichia coli strain 536 (O6:K15:H31) carries two alpha-haemolysin determinants which are located on different pathogenicity islands (PAI ISUB536 and PAI IISUB536). PAI IISUB536 is associated with the tRNA gene leuX. The leuX-encoded tRNASUB5SUPLeu is required for the efficient expression of the hly determinants in strain 536. HlyA levels were reduced and secretion of the protein was delayed in the leuX

-negative mutant strain 536DELTA102. The lack of a functional tRNASUB5SUPLeu resulted in a decrease in hly transcript levels in comparison to the wild-type strain. Analysis of several genes whose products are involved in the regulation of hly expression revealed that
levels of RfaH and Hha, as well as the corresponding rfaH and hha transcripts, were higher in the leuX-negative background, whereas the expression of tolC and hns was not influenced by the leuX genotype. The analysis of hly transcript levels in hha deletion mutants of the E. coli strains 536 and 536DELTA102 demonstrated that the increase in hha
expression is partially responsible for the reduction in hly transcript
levels in the leuX-negative background. These results demonstrate that the tRNASUB5SUPLeu affects the expression of the alpha-haemolysin determinant at different levels in a regulatory cascade, and imply that, in
addition to Hha, at least one further, as yet unidentified, regulatory factor must be involved in the regulation of hly transcription in the uropathogenic E. coli strain 536.

17/7/48 (Item 6 from file: 71)
DIALOG(R)File 71:ELSEVIER BIOBASE

01975667 2002056614
Recombinogenic effects of suppressors of position-effect variegation in
Drosophila
Westphal T.; Reuter G.
ADDRESS: G. Reuter, Institute of Genetics, Martin Luther University, D-06120 Halle, Weinbergweg 10, Germany
EMAIL: reuter@genetik.uni-halle.de
Journal: Genetics, 160/2 (609-621), 2002, United States
CODEN: GENTA
ISSN: 0016-6731
DOCUMENT TYPE: Article
LANGUAGES: English SUMMARY LANGUAGES: English
NO. OF REFERENCES: 68

Compact chromatin structure, induction of gene silencing in position-effect
variegation (PEV), and crossing-over suppression are typical features of
heterochromatin. To identify genes affecting crossing-over suppression by
heterochromatin we tested PEV suppressor mutations for their effects on crossing over in pericentromeric regions of Drosophila autosomes. From
the 46 mutations (28 loci) studied, 16 Su(var) mutations of the nine genes Su(var)2-1, Su(var)2-2, Su(var)2-5, Su(var)2-10, Su(var)2-14,

Su(var) 2-15, Su(var)3-3, Su(var)3-7, and Su(var)3-9 significantly increase
in heterozygotes or by additive effects in double and triple heterozygotes
crossing over in the rip-SUPp region of chromosome 3. Su(var)2-2SUP01
and Su(var) 2-14SUP01 display the strongest recombinogenic effects and were
also shown to enhance recombination within the light-rolled heterochromatic
region of chromosome 2. The dominant recombinogenic effects of Su(var)
mutations are most pronounced in proximal euchromatin and are
accompanied with significant reduction of meiotic nondisjunction. Our data
suggest that crossing-over suppression by heterochromatin is controlled at
chromatin structure as well as illustrate the possible effects of
heterochromatin on total crossing-over frequencies in the genome.

17/7/49 (Item 7 from file: 71)
DIALOG(R)File 71:ELSEVIER BIOBASE

01421702 2000094567
Influence of pathogenicity islands and the minor leuX-encoded
tRNA$D5(Leu) on the proteome pattern of the uropathogenic Escherichia
coli strain 536
Piechaczek K.; Dobrindt U.; Schierhorn A.; Fischer G.S.; Hecker M.; Hacker
J.
ADDRESS: Dr. J. Hacker, Inst. fur Molekul. Infektionsbiol., Rontgenring 11,
D-97070 Wurzburg, Germany
EMAIL: j.hacker@mail.uni-wuerzburg.de
Journal: International Journal of Medical Microbiology, 290/1 (75-84), 2000
, Germany
CODEN: IMEMF
ISSN: 1438-4221
DOCUMENT TYPE: Article
LANGUAGES: English SUMMARY LANGUAGES: English
NO. OF REFERENCES: 37

The uropathogenic Escherichia coli strain 536 (O6:K15:H31) carries four
distinct DNA regions in its chromosome, termed pathogenicity islands (PAIs
I$D5inf 3inf 6 to IV$D5inf 3inf 6). Each of these PAIs encodes at least one
virulence factor. All four PAIs are associated with tRNA genes. PAI I$D5inf

3inf 6 and PAI II$D5inf 3inf 6 can be spontaneously deleted from the chromosome by homologous recombination between flanking direct repeats. The deletion of PAI II$D5inf 3inf 6 results in the truncation of the associated gene leuX encoding the tRNA$D5(Leu). This tRNA influences the expression of various virulence traits. In order to get a deeper insight into the role of PAI I$D5inf 3inf 6/II$D5inf 3inf 6 and of the tRNA$D5(Leu) for the protein expression, the protein expression patterns of Escherichia coli 536 and different derivatives were studied. Differences in the protein expression patterns of the wild-type strain Escherichia coli 536, its mutants 536-21 (PAI I$D5inf 3inf 6sup -, PAI II$D5inf 3inf 6sup -, leuXsup -), 536Delta102 (PAI I$D5inf 3inf 6sup +, PAI II$D5inf 3inf 6sup +, leuXsup -) as well as of the strain 536R3 (PAI I$D5inf 3inf 6sup -, PAI II$D5inf 3inf 6sup -, leuXsup +) were analyzed by two-dimensional polyacrylamide gel electrophoresis and MALDI-TOF mass spectrometry. We identified about 39 different intracellular proteins whose expression is markedly altered in the different strain backgrounds. These differences can be linked either to the presence or absence of the PAI I$D5inf 3inf 6 and PAI II$D5inf 3inf 6 or to that of the tRNA gene leuX. The identities of 34 proteins have been determined by MALDI-TOF-MS. The identification of five proteins was not possible. The results suggest that proteome analysis is an efficient approach to study differences in global gene expression.
The comparison of protein expression patterns of the uropathogenic E. coli strain 536 and different derivatives revealed that in this strain the expression of various proteins including those encoded by many housekeeping genes is affected by the presence of PAI I$D5inf 3inf 6 and Pai II$D5inf 3inf 6 or by that of the tRNA$D5(Leu).

01086882 1998110253
The leuX-encoded tRNA$D5(Leu) but not the pathogenicity islands I and II influence the survival of the uropathogenic Escherichia coli strain

536 in CD-1 mouse bladder mucus in the stationary phase
Dobrindt U.; Cohen P.S.; Utley M.; Muhldorfer I.; Hacker J.
ADDRESS: J. Hacker, Inst. fur Mole. Infektionsbiologie, Universitat Wurzburg, Rontgenring 11, 97070 Wurzburg, Germany
EMAIL: j.hacker@rzbox.uni-wuerzburg.de
Journal: FEMS Microbiology Letters, 162/1 (135-141), 1998, Netherlands
PUBLICATION DATE: May 1, 1998
CODEN: FMLED
ISSN: 0378-1097
PUBLISHER ITEM IDENTIFIER: S0378109798001141
DOCUMENT TYPE: Article
LANGUAGES: English SUMMARY LANGUAGES: English
NO. OF REFERENCES: 22

The uropathogenic Escherichia coil strain 536 carries two pathogenicity islands, each of which is associated with either of the tRNA genes selC or leuX, respectively. Growth competition in CD-1 mouse mucus between the wild- type strain E. coli 536, its leuX mutant 536Delta102 and its mutant 536R3, lacking both pathogenicity islands but expressing a functional tRNA$D5(Leu), revealed a major impact of leuX on E. coli survival in bladder mucus. The impaired survival in CD-1 mouse mucus observed upon deletion of the leuX gene was abolished after complementation with the leuX gene. The survival of bacteria in bladder mucus was not influenced by the presence of pathogenicity islands I and II.

17/7/51 (Item 9 from file: 71)
DIALOG(R)File 71:ELSEVIER BIOBASE

01074126 1999062550
Novel temperature-sensitive mutants of Escherichia coli that are unable to grow in the absence of wild-type tRNAinf 6/(Leu)
Nakayashiki T.; Inokuchi H.
ADDRESS: H. Inokuchi, Department of Biophysics, Faculty of Science, Kyoto University, Sakyo-ku, Kyoto 606-8502, Japan
EMAIL: 00hachi@molbio.biophys.kyoto-u.ac.jp
Journal: Journal of Bacteriology, 180/11 (2931-2935), 1998, United States
CODEN: JOBAA
ISSN: 0021-9193
DOCUMENT TYPE: Article
LANGUAGES: English SUMMARY LANGUAGES: English
NO. OF REFERENCES: 27

Escherichia coli has only a single copy of a gene for tRNAinf 6/(Leu) (Y.

Komine et al., J. Mol. Biol. 212:579-598, 1990). The anticodon of this tRNA
is CAA (the wobble position C is modified to Osup 2-methylcytidine), and it
recognizes the codon UUG. Since UUG is also recognized by tRNAinf 4/(Leu),
which has UAA (the wobble position U is modified to
5-carboxymethylaminomethyl- Osup 2-methyluridine) as its anticodon, tRNAinf
6/(Leu) is not essential for protein synthesis. The BT63 strain has a
mutation in the anticodon of tRNAinf 6/(Leu) with a change from CAA
to CUA, which results in the amber suppressor activity of this strain (
supP, Susup +6). We isolated 18 temperature- sensitive (ts) mutants
of the BT63 strain whose temperature sensitivity was complemented by
introduction of the wild-type gene for tRNAinf 6/(Leu). These tRNAinf
6/(Leu)-requiring mutants were classified into two groups. The 10 group I
mutants had a mutation in the miaA gene, whose product is involved in
a modification of tRNAs that stabilizes codon-anticodon interactions.
Overexpression of the gene for tRNAinf 4/(Leu) restored the growth of group
I mutants at 42degreeC. Replacement of the CUG codon with UUG reduced the
efficiency of translation in group I mutants. These results suggest that
unmodified tRNAinf 4/(Leu) poorly recognizes the UUG codon at 42degreeC and
that the wild-type tRNAinf 6/(Leu) is required for translation in order to
maintain cell viability. The mutations in the six group II mutants
were complemented by introduction of the gidA gene, which may be involved
in cell division. The reduced efficiency of translation caused by
replacement of the CUG codon with UUG was also observed in group II
mutants. The mechanism of requirement for tRNAinf 6/(Leu) remains to be
investigated.

00703127 97207820
The Pai-associated leuX specific tRNA$D5(Leu) affects type 1
fimbriation in pathogenic Escherichia coli by control of FimB recombinase
expression
Ritter A.; Gally D.L.; Olsen P.B.; Dobrindt U.; Friedrich A.; Klemm P.;
Hacker J.

ADDRESS: J. Hacker, Inst. fur Mol. Infektionsbiologie, Rontgenring 11,
D-97070 Wurzburg, Germany
EMAIL: j.hacker@rzbox.uni-wuerzburg.de
Journal: Molecular Microbiology, 25/5 (871-882), 1997, United Kingdom
PUBLICATION DATE: 19970000
CODEN: MOMIE
ISSN: 0950-382X
DOCUMENT TYPE: Article
LANGUAGES: English SUMMARY LANGUAGES: English
NO. OF REFERENCES: 52

The uropathogenic Escherichia coli strain 536 (O6:K15: H31) carries two
large chromosomal pathogenicity islands (Pais). Both Pais are flanked by
tRNA genes. Spontaneous deletion of pai II results in truncation of
the leuX tRNA$D5(Leu) gene. This tRNA is required for the expression
of type 1 fimbriae (Fim) and other virulence factors. Transcription of
fimA, encoding the major type 1 fimbrial subunit is controlled by an
invertable DNA switch. The inversion is catalysed by two recombinases, FimB
and FimE. FimB is able to turn the switch on, FimE only off. The fimB gene
of strain 536 contains five TTG codons recognized by tRNA$D5(Leu) fimE
contains only two. It was proposed that turning on the fim switch requires
efficient translation of FimB, in turn requiring tRNA$D5(Leu). Strains in
which the TTG codons in fimB were replaced with CTG codons at the wild-type
locus were able to produce type 1 fimbriae in the absence of leuX.
fimB transcription was influenced by the presence of leuX, but only
slightly affected by the presence or absence of leuX codons in fimB.
FimB translation was significantly higher from codon-replaced fimB genes
than that of wild-type fimB genes in various strain backgrounds. The fim
switch was shown to be switched off in leuXsup - derivatives of E. coli
536, but could be found in the on position when the codon-altered fimB gene
was exchanged into the chromosome of these strains. From these data, it is
apparent that tRNA$D5(Leu) is required for efficient translation of FimB,
in turn, leading to type 1 fimbrial expression.

0079032913 EMBASE No: 2002196621

Efficient expression of the alpha-haemolysin determinant in the uropathogenic Escherichia coli strain 536 requires the leuX-encoded tRNA SUB 5 SUP Leu

Dobrindt U.; Emody L.; Gentschev I.; Goebel W.; Hacker J.
Institut fur Molekulare Infektionsbiologie, Universitat Wurzburg, Rontgenring 11, 97070 Wurzburg, Germany
AUTHOR EMAIL: j.hacker@mail.uni-wuerzburg.de
CORRESP. AUTHOR/AFFIL: Hacker J.: Inst. fur Molek. Infektionsbiologie, Universitat Wurzburg, Rontgenring 11, 97070 Wurzburg, Germany
CORRESP. AUTHOR EMAIL: j.hacker@mail.uni-wuerzburg.de

Molecular Genetics and Genomics (Mol. Genet. Genomics) (Germany) June 15, 2002, 267/3 (370-379)
CODEN: MGGOA ISSN: 1617-4615
DOI: 10.1007/s00438-002-0668-3
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 43

The uropathogenic Escherichia coli strain 536 (O6:K15:H31) carries two alpha-haemolysin determinants which are located on different pathogenicity islands (PAI I SUB 536 and PAI II SUB 536). PAI II SUB 536 is associated with the tRNA gene leuX. The leuX-encoded tRNA SUB 5 SUP Leu is required for the efficient expression of the hly determinants in strain 536. HlyA levels were reduced and secretion of the protein was delayed in the leuX-negative mutant strain 536Delta102. The lack of a functional tRNA SUB 5 SUP Leu resulted in a decrease in hly transcript levels in comparison to the wild-type strain. Analysis of several genes whose products are involved in the regulation of hly expression revealed that levels of RfaH and Hha, as well as the corresponding rfaH and hha transcripts, were higher in the leuX-negative background, whereas the expression of tolC and hns was not influenced by the leuX genotype. The analysis of hly transcript levels in hha deletion mutants of the E. coli strains 536 and 536Delta102 demonstrated that the increase in hha expression is partially responsible for the reduction in hly transcript levels in the leuX-negative background. These results demonstrate that the tRNA SUB 5 SUP Leu affects the expression of the alpha-haemolysin determinant at different levels in a regulatory cascade, and imply that, in addition to Hha, at least one further, as yet unidentified, regulatory

factor must be involved in the regulation of hly transcription in the uropathogenic E. coli strain 536.

17/7/54 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

0077608023 EMBASE No: 1999094182
Novel temperature-sensitive mutants of Escherichia coli that are unable
to grow in the absence of wild-type tRNA SUB 6/(Leu)
Nakayashiki T.; Inokuchi H.
Department of Biophysics, Faculty of Science, Kyoto University, Sakyo-ku,
Kyoto 606-8502, Japan
AUTHOR EMAIL: 00hachi@molbio.biophys.kyoto-u.ac.jp
CORRESP. AUTHOR/AFFIL: Inokuchi H.: Department of Biophysics, Faculty of
Science, Kyoto University, Sakyo-ku, Kyoto 606-8502, Japan
CORRESP. AUTHOR EMAIL: 00hachi@molbio.biophys.kyoto-u.ac.jp

Journal of Bacteriology (J. Bacteriol.) (United States) June 1, 1998,
180/11 (2931-2935)
CODEN: JOBAA ISSN: 0021-9193
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 27

Escherichia coli has only a single copy of a gene for tRNA SUB 6/(Leu)
(Y. Komine et al., J. Mol. Biol. 212:579-598, 1990). The anticodon of this
tRNA is CAA (the wobble position C is modified to O SUP 2-methylcytidine),
and it recognizes the codon UUG. Since UUG is also recognized by tRNA SUB
4/(Leu), which has UAA (the wobble position U is modified to
5-carboxymethylaminomethyl- O SUP 2-methyluridine) as its anticodon, tRNA
SUB 6/(Leu) is not essential for protein synthesis. The BT63 strain has a
mutation in the anticodon of tRNA SUB 6/(Leu) with a change from CAA to CUA, which results in the amber suppressor activity of this strain (
supP, Su SUP +6). We isolated 18 temperature- sensitive (ts) mutants of the BT63 strain whose temperature sensitivity was complemented by introduction of the wild-type gene for tRNA SUB 6/(Leu). These tRNA SUB
6/(Leu)-requiring mutants were classified into two groups. The 10 group I

mutants had a mutation in the miaA gene, whose product is involved in a modification of tRNAs that stabilizes codon-anticodon interactions. Overexpression of the gene for tRNA SUB 4/(Leu) restored the growth of group I mutants at 42(deg)C. Replacement of the CUG codon with UUG reduced
the efficiency of translation in group I mutants. These results suggest
that unmodified tRNA SUB 4/(Leu) poorly recognizes the UUG codon at 42(deg)C and that the wild-type tRNA SUB 6/(Leu) is required for translation in order to maintain cell viability. The mutations in the six group II mutants were complemented by introduction of the gidA gene,
which may be involved in cell division. The reduced efficiency of translation caused by replacement of the CUG codon with UUG was also observed in group II mutants. The mechanism of requirement for tRNA SUB
6/(Leu) remains to be investigated.

17/7/55 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE

0077250110 EMBASE No: 1998160268
The leuX-encoded tRNA SUB 5(Leu) but not the pathogenicity islands I and II influence the survival of the uropathogenic Escherichia coli strain 536 in CD-1 mouse bladder mucus in the stationary phase
Dobrindt U.; Cohen P.S.; Utley M.; Muhldorfer I.; Hacker J.
Inst. fur Molec. Infektionsbiologie, Universitat Wurzburg, Rontgenring
11, 97070 Wurzburg, Germany
AUTHOR EMAIL: j.hacker@rzbox.uni-wuerzburg.de
CORRESP. AUTHOR/AFFIL: Hacker J.: Inst. fur Mole. Infektionsbiologie,
Universitat Wurzburg, Rontgenring 11, 97070 Wurzburg, Germany
CORRESP. AUTHOR EMAIL: j.hacker@rzbox.uni-wuerzburg.de

FEMS Microbiology Letters (FEMS Microbiol. Lett.) (Netherlands) May 1,
1998, 162/1 (135-141)
CODEN: FMLED ISSN: 0378-1097
PUBLISHER ITEM IDENTIFIER: S0378109798001141
DOI: 10.1016/S0378-1097(98)00114-1
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 22
The uropathogenic Escherichia coil strain 536 carries two pathogenicity
islands, each of which is associated with either of the tRNA genes selC or
leuX, respectively. Growth competition in CD-1 mouse mucus between the wild- type strain E. coli 536, its leuX mutant 536Delta102 and

its mutant 536R3, lacking both pathogenicity islands but expressing a functional tRNA SUB 5(Leu), revealed a major impact of leuX on E. coli survival in bladder mucus. The impaired survival in CD-1 mouse mucus
observed upon deletion of the leuX gene was abolished after complementation with the leuX gene. The survival of bacteria in bladder mucus was not influenced by the presence of pathogenicity islands I
and II.

17/7/56 (Item 4 from file: 73)
DIALOG(R)File 73:EMBASE

0076992778 EMBASE No: 1997285998
The Pai-associated leuX specific tRNA SUB 5(Leu) affects type 1 fimbriation in pathogenic Escherichia coli by control of FimB recombinase
expression
Ritter A.; Gally D.L.; Olsen P.B.; Dobrindt U.; Friedrich A.; Klemm P.;
Hacker J.
Inst. F. Molec. Infektionsbiologie, Rontgenring 11, D-97070 Wurzburg,
Germany
AUTHOR EMAIL: j.hacker@rzbox.uni-wuerzburg.de
CORRESP. AUTHOR/AFFIL: Hacker J.: Inst. fur Mol. Infektionsbiologie, Rontgenring 11, D-97070 Wurzburg, Germany

Molecular Microbiology (MOL. MICROBIOL.) (United Kingdom) October 2,
1997, 25/5 (871-882)
CODEN: MOMIE ISSN: 0950-382X
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 52

The uropathogenic Escherichia coli strain 536 (O6:K15: H31) carries two
large chromosomal pathogenicity islands (Pais). Both Pais are flanked by
tRNA genes. Spontaneous deletion of pai II results in truncation of the leuX tRNA SUB 5(Leu) gene. This tRNA is required for the expression of type 1 fimbriae (Fim) and other virulence factors. Transcription of fimA, encoding the major type 1 fimbrial subunit is controlled by an invertable DNA switch. The inversion is catalysed by two
recombinases, FimB and FimE. FimB is able to turn the switch on, FimE only
off. The fimB gene of strain 536 contains five TTG codons recognized by

tRNA SUB 5(Leu) fimE contains only two. It was proposed that turning on the
fim switch requires efficient translation of FimB, in turn requiring tRNA
SUB 5(Leu). Strains in which the TTG codons in fimB were replaced with CTG
codons at the wild-type locus were able to produce type 1 fimbriae in the
absence of leuX. fimB transcription was influenced by the presence of
leuX, but only slightly affected by the presence or absence of
leuX codons in fimB. FimB translation was significantly higher from
codon-replaced fimB genes than that of wild-type fimB genes in various
strain backgrounds. The fim switch was shown to be switched off in
leuX SUP - derivatives of E. coli 536, but could be found in the on
position when the codon-altered fimB gene was exchanged into the chromosome
of these strains. From these data, it is apparent that tRNA SUB 5(Leu) is
required for efficient translation of FimB, in turn, leading to type 1
fimbrial expression.

17/7/57 (Item 5 from file: 73)
DIALOG(R)File 73:EMBASE

0073267668 EMBASE No: 1986031702
Identification of transfer RNA suppressors in Escherichia coli. IV. Amber
suppressor Su SUP +6 a double mutant of a new species of leucine tRNA
Yoshimura M.; Inokuchi H.; Ozeki H.
Department of Biophysics, Faculty of Science, Kyoto University, Kyoto 606
, Japan:
CORRESP. AUTHOR/AFFIL: Department of Biophysics, Faculty of Science, Kyoto University, Kyoto 606, Japan

Journal of Molecular Biology (J. MOL. BIOL.) (United Kingdom) December
1, 1984, 177/4 (627-644)
CODEN: JMOBA ISSN: 0022-2836
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English

An Escherichia coli DNA fragment containing an Su SUP +6 amber suppressor
gene (supP) was cloned into a lambdagtlambdaCh vector by the shotgun
method, selecting a Su SUP +6 transducing phage lambdapSu SUP +6. Through
prophage integration followed by induction occurring at the transducing
region of the lambdapSu SUP +6 in Su SUP - E. coli, a counterpart

transducing phage carrying the wild-type allele (Su(deg)6) was isolated
(lambdapSu(deg)6). The fingerprint of a tRNA coded by lambdapSU(deg)6 was
identical to that of an unindentified tRNA(E) previously reported (Ikemura
& Ozeki, 1977). The cloverleaf structure of this tRNA was determined by
combining the results of tRNA analysis and DNA sequencing of the gene.
Judging from the anticodon of 5'-CAA'3', Su(deg)6 tRNA was identified as a
new type of leucine isoacceptor in E. coli. Unlike other suppressors
analyzed, Su SUP +6 tRNA differed by two nucleotides fromSu(deg)6 tRNA; one
at the anticodon (CAA to CUA) and the other at the junction of D- and
anticodon-stem (A27 to G27). DNA sequence analysis revealed that a single
stretch of tRNA is flanked by the putative sequences of promoter and
terminator. Thus a single copy of the Su(deg)6 tRNA gene constitutes a
solitary tRNA transcription unit. Southern blotting showed only one copy of
Su(deg)6 tRNA gene per haploid genome of E. coli. Since this single gene
can mutate to the Su SUP +6 suppressor, the Su(deg)6 leucine tRNA may
be accounted as a dispensable specis among the leucine isoacceptor tRNAs.
Two possible open reading frames are found immediately following the
Su(deg)6 tRNA gene.

0072626367 EMBASE No: 1984156782
Purkinye cell activity in the primate flocculus during optokinetic
stimulation, smooth pursuit eye movements and VOR-suppression
Buttner U.; Waespe W.
Department of Neurology, University of Zurich, CH-8091 Zurich,
Switzerland:
CORRESP. AUTHOR/AFFIL: Department of Neurology, University of Zurich,
CH-8091 Zurich, Switzerland

Experimental Brain Research (EXP. BRAIN RES.) (Germany) August 23,
1984, 55/1 (97-104)
CODEN: EXBRA ISSN: 0014-4819
DOCUMENT TYPE: Journal RECORD TYPE: Abstract
LANGUAGE: English

Purkinje cell (PC) activity in the flocculus of trained monkeys was

recorded during: 1) Vestibular stimulation in darkness. 2) Suppression of
the vestibulo-ocular reflex (VOR-supp) by fixation of a small light
spot stationary with respect to the monkey. 3) Visual-vestibular conflict
(i.e. the visual surround moves together with the monkey during vestibular
stimulation), which leads to attenuation or suppression of vestibular
nystagmus. 4) Smooth pursuit eye movements. 5) Optokinetic nystagmus (OKN).
6) Suppression of nystagmus during optokinetic stimulation (OKN-supp)
by fixation of a small light spot; whereby stimulus velocity corresponds
then to image slip velocity. Results were obtained from PCs, which were
activated with VOR-supp during rotation to the ipsilateral side. The
same PCs were also modulated during smooth pursuit and visual-vestibular
conflict. No tonic modulation during constant velocity OKN occurred with
slow-phase nystagmus velocities below 4-60 deg/s. Tonic responses were only
seen at higher nystagmus velocities. Transient activity changes appeared at
the beginning and end of optokinetic stimulation. PCs were not modulated by
image slip velocity during OKN-supp. The results show that in
primates the same population of floccular PCs is involved in different
mechanisms of visual-vestibular interaction and that smooth pursuit and
certain components of OKN slow-phase velocity share the same neural
pathway. It is argued that the activity of these neurons can neither be
related strictly to gaze, eye or image slip velocity; instead, their
activity pattern can be best interpreted by assuming a modulation, which is
complementary to that of central vesibular neurons of the vestibular
nuclei, in the control of slow eye movements.

0071414055 EMBASE No: 1979146374
Suppression of cytotoxic response to histoincompatible cells. II. Analysis of the role of two independent T suppressor pools in maintenance
of neonatally induced allograft tolerance in mice
Gorczynski R.M.; MacRae S.
Ontario Cancer Inst., Toronto, Canada:
CORRESP. AUTHOR/AFFIL: Ontario Cancer Inst., Toronto, Canada

Journal of Immunology (J. IMMUNOL.) (United States) July 23, 1979,
122/3 (747-752)
CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English

The kinetics of appearance of the precursors of Supp(A) cells (capable of inhibiting CTL(p)-->CTL) or Supp(B) cells (capable of inhibiting (stem cells -->CTL(p)) in neonatal mice, as well as the appearance of Supp(A)/Supp(B) cells in mice given neonatal innoculations of semiallogeneic spleen cells has been investigated. The data obtained are consistent with the idea that Supp(A) cells have a natural role to play in the induction of neonatal tolerance, whereas Supp(B) cells may be more important for the maintenance of the tolerant state. Unlike the level of Supp(B) cells, the level of Supp(A) cells in tolerant mice seems to be modulated by the presence of the tolerizing determinants. Data are provided to show that Supp (B) cells, once induced in tolerant mice, can adoptively transfer specific allograft unresponsiveness to newborn syngeneic mice in the absence of added tolerizing antigen, whereas Supp(A) cells are not able to do so. These data fit the notion that Supp(B) cells may be responsible for the phenotype of clonal deletion.

0000158441 IP ACCESSION NO: 6244514

Investigating metabolite essentiality through genome-scale analysis of Escherichia coli production capabilities

Imielinski, Marcin; Belta, Calin; Halasz, Adam; Rubin, Harvey
Genomics and Computational Biology Graduate Group, University of Pennsylvania School of Medicine Philadelphia, PA, 19104, USA. Department of Medicine, University of Pennsylvania School of Medicine Philadelphia, PA, 19104, USA. Department of Mechanical Engineering, Drexel University Philadelphia, PA, 19104, USA. General Robotics, Automation, Sensing and Perception Laboratory, University of Pennsylvania Philadelphia, PA, 19104, USA

Bioinformatics, v 21, n 9, p 2008-2016, May 1, 2005
PUBLICATION DATE: 2005

PUBLISHER: Oxford University Press, Oxford Journals, Great Clarendon Street
Oxford OX2 6DP UK, [mailto:jnl.samples@oup.co.uk],
[URL:http://www3.oup.co.uk/jnls/]

DOCUMENT TYPE: Journal Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English
ISSN: 1367-4803
ELECTRONIC ISSN: 1460-2059
FILE SEGMENT: BioEngineering Abstracts

ABSTRACT:
MOTIVATION: A phenotype mechanism is classically derived through the study of a set of mutants and comparison of their biochemical capabilities.
One method of comparing mutant capabilities is to characterize producible
and knocked out metabolites. However such an effect is difficult to manually assess, especially for a large biochemical network and a complex
media. Current algorithmic approaches towards analyzing metabolic networks
either do not address this specific property or are computationally infeasible on the genome-scale. RESULTS: We have developed a novel genome-scale computational approach that identifies the full set of biochemical species that are knocked out from the metabolome following a
gene deletion. Results from this approach are combined with data from in vivo mutant screens to examine the essentiality of metabolite production
for a phenotype. This approach can also be a useful tool for metabolic network annotation validation and refinement in newly sequenced organisms.
Combining an in silico genome-scale model of Escherichia coli metabolism
with in vivo survival data, we uncover possible essential roles for several
cell membranes, cell walls, and quinone species. We also identify specific
biomass components whose production appears to be non-essential for survival, contrary to the assumptions of previous models.
AVAILABILITY:
Programs are available upon request from the authors in the form of Matlab
script files. CONTACT: imielns[at]mail.med.upenn.edu Supplementary information:
http://www.cis.upenn.edu/biocomp/manuscripts/bioinformatics
bti245 / supp-info.html

17/7/61 (Item 1 from file: 144)
DIALOG(R)File 144:Pascal

17236738 PASCAL Number: 05-0308420

Multiple insertional events, restricted by the genetic background, have led to acquisition of pathogenicity island II SUB J SUB 9 SUB 6 -like domains among Escherichia coli strains of different clinical origins

BIDET Philippe; BONACORSI Stephane; CLERMONT Olivier; DE MONTILLE Caroline; BRAHIMI Naima; BINGEN Edouard

Laboratoire d'etudes de genetique bacterienne dans les infections de l'enfant (EA3105), Universite Denis Diderot-Paris 7, Service de Microbiologie, Hopital Robert Debre (AP-HP), 75019 Paris, France

Journal: Infection and immunity, 2005, 73 (7) 4081-4087

ISSN: 0019-9567 CODEN: INFIBR Availability: INIST-15757; 354000138518200290

Number of Refs.: 41 reference

Document Type: P (Serial) ; A (Analytic)

Country of Publication: United States

Language: English

We investigated the dissemination of pathogenicity island (PAI) II SUB J SUB 9 SUB 6 -like elements (hra, hly, cnfl, and pap) among 455 Escherichia coli isolates from children and adults with urinary tract infection (UTI), neonates with meningitis or colonized healthy neonates, and 74 reference strains by means of PCR phylogenetic grouping, ribotyping, and PCR analysis of virulence genes. Colocalization of these genes was documented by pulsed-field gel electrophoresis followed by Southern hybridization and long-range PCR (LRPCR) between the hra and the papG alleles. Site-specific insertion of the PAI was determined by LRPCR between hra and tRNA flanking sequences. hra, hly, and cnfl were found in 113 isolates and consistently colocalized, constituting the backbone of PAI II SUB J SUB 9 SUB 6 -like domains. The prevalence of PAI II SUB J SUB 9 SUB 6 -like domains was significantly higher among UTI isolates than among neonatal meningitis and commensal isolates. These domains were restricted to a few ribotypes of group B2. In contrast to the consistent colocalization of hra, hly, and cnfl, the pap operon was varied: 12% of strains exhibited an allelic

exchange of the papG class III allele (papGIII) for the papG class II allele (papGII) (only UTI isolates), and the pap operon was deleted in 23% of strains. No strains harbored papGIII outside the PAI, which appears to be the only source of this allele. PAI II SUB J SUB 9 SUB 6 -like domains were inserted in the vicinities of three different tRNAs-pheU (54%), leuX (29%), and pheV (15%)-depending on the genetic backgrounds and origins of the isolates. Multiple insertional events restricted by the genetic background have thus led to PAI II SUB J SUB 9 SUB 6 acquisition. Specific genetic backgrounds and insertion sites may have played a role in additional recombination processes for E. coli adaptation to different ecological niches.

17/7/62 (Item 2 from file: 144)
DIALOG(R)File 144:Pascal

14673396 PASCAL Number: 00-0346817
Influence of pathogenicity islands and the minor leuX-encoded tRNA SUB 5 SUP L SUP e SUP u on the proteome pattern of the uropathogenic Escherichia coli strain 536
PIECHACZEK K; DOBRINDT U; SCHIERHORN A; FISCHER G S; HECKER M; HACKER J
Institut fuer Molekulare Infektionsbiologie, Roentgenring 11, 97070 Wuerzburg, Germany; Forschungsstelle Enzymologie der Proteinfaltung der Max-Planck-Gesellschaft, Weinbergweg 22, D-06120 Halle/Saale and Martin-Luther-Universitaet, Fachbereich Biochemie, Biotechnologie, 06099 Halle (Saale), Germany; Institut fuer Mikrobiologie und Molekularbiologie, Universitaet Greifswald, F.-L.-Jahn-Str. 15, 17487 Greifswald, Germany
Journal: IJMM. International journal of medical microbiology, 2000, 290 (1) 75-84
ISSN: 1438-4221 Availability: INIST-3329; 354000086952320080
Number of Refs.: 1 p.1/4
Document Type: P (Serial) ; A (Analytic)
Country of Publication: Germany
Language: English
The uropathogenic Escherichia coli strain 536 (O6:K15:H31) carries four distinct DNA regions in its chromosome, termed pathogenicity islands (PAIs

I SUB 5 SUB 3 SUB 6 to IV SUB 5 SUB 3 SUB 6). Each of these PAIs encodes at least one virulence factor. All four PAIs are associated with tRNA genes. PAI I SUB 5 SUB 3 SUB 6 and PAI II SUB 5 SUB 3 SUB 6 can be spontaneously deleted from the chromosome by homologous recombination between flanking direct repeats. The deletion of PAI II SUB 5 SUB 3 SUB 6 results in the truncation of the associated gene leuX encoding the tRNA SUB 5 SUP L SUP e SUP u . This tRNA influences the expression of various virulence traits. In order to get a deeper insight into the role of PAI I SUB 5 SUB 3 SUB 6 /II SUB 5 SUB 3 SUB 6 and of the tRNA SUB 5 SUP L SUP e SUP u for the protein expression, the protein expression patterns of Escherichia coli 536 and different derivatives were studied. Differences in the protein expression patterns of the wild-type strain Escherichia coli 536, its mutants 536-21 (PAI I SUB 5 SUB 3 SUB 6 SUP - , PAI II SUB 5 SUB 3 SUB 6 SUP - , leuX SUP -), 536A102 (PAI I SUB 5 SUB 3 SUB 6 SUP + , PAI II SUB 5 SUB 3 SUB 6 SUP + , leuX SUP -) as well as of the strain 536R3 (PAI I SUB 5 SUB 3 SUB 6 SUP - , PAI II SUB 5 SUB 3 SUB 6 SUP - , leuX SUP +) were analyzed by two-dimensional polyacrylamide gel electrophoresis and MALDI-TOF mass spectrometry. We identified about 39 different intracellular proteins whose expression is markedly altered in the different strain backgrounds. These differences can be linked either to the presence or absence of the PAI I SUB 5 SUB 3 SUB 6 and PAI II SUB 5 SUB 3 SUB 6 or to that of the tRNA gene leuX. The identities of 34 proteins have been determined by MALDI-TOF-MS. The identification of five proteins was not possible. The results suggest that proteome analysis is an efficient approach to study differences in global gene expression. The comparison of protein expression patterns of the uropathogenic E. coli strain 536 and different derivatives revealed that in this strain the expression of various proteins including those encoded by many housekeeping genes is affected by the presence of PAI I SUB 5 SUB 3 SUB 6 and Pai II SUB 5 SUB 3 SUB 6 or by that of the tRNA SUB 5 SUP L SUP e SUP u .

17/7/63 (Item 3 from file: 144)
DIALOG(R)File 144:Pascal

13624427 PASCAL No.: 98-0330446
The leuX-encoded tRNA SUB 5 SUP L SUP e SUP u but not the pathogenicity islands I and II influence the survival of the uropathogenic
Escherichia coli strain 536 in CD-1 mouse bladder mucus in the stationary
phase
DOBRINDT U; COHEN P S; UTLEY M; MUEHLDORFER I; HACKER J
Institut fuer Molekulare Infektionsbiologie, Universitaet Wuerzburg, Roentgenring 11, 97070 Wuerzburg, Germany; Department of Biochemistry, Microbiology and Molecular Genetics, University of Rhode Island, Kingston,
RI 02881, United States
Journal: FEMS microbiology letters, 1998, 162 (1) 135-141
ISSN: 0378-1097 CODEN: FMLED7 Availability: INIST-17567A; 354000075599340200
No. of Refs.: 22 ref.
Document Type: P (Serial) ; A (Analytic)
Country of Publication: Netherlands
Language: English
The uropathogenic Escherichia coli strain 536 carries two pathogenicity
islands, each of which is associated with either of the tRNA genes selC or
leuX , respectively. Growth competition in CD-1 mouse mucus between the wild-type strain E. coli 536, its leuX mutant 536 DELTA 102 and its mutant 536R3, lacking both pathogenicity islands but expressing a
functional tRNA, SUP L SUP e SUP u revealed a major impact of leuX on E. coli survival in bladder mucus. The impaired survival in CD-I mouse
mucus observed upon deletion of the leuX gene was abolished after complementation with the leuX gene. The survival of bacteria in bladder mucus was not influenced by the presence of pathogenicity islands I
and II.

17/7/64 (Item 4 from file: 144)
DIALOG(R)File 144:Pascal

12560537 PASCAL No.: 96-0242892
Remodelage du code genetique chez Escherichia coli
(Remodelling the genetic code in Escherichia coli)
LEMEIGNAN Beatrice; LACROUTE F, dir
Universite de Paris 06, Paris, Francee
Univ.: Universite de Paris 06. Paris. FRA Degree: Th. doct. 1995-02; 1995 148 p.
Availability: INIST-T 103086; T95PA066140
No. of Refs.: 175 ref.
Document Type: T (Thesis) ; M (Monographic)
Country of Publication: France
Language: French Summary Language: French; English
La constitution chimique des proteines semble restreinte aux combinaisons d'un meme jeu de vingt acides amines chez tous les etres vivants connus. Cette invariance, fondee sur l'extreme conservation des processus de la traduction genetique, n'implique neanmoins nullement que l'elargissement du jeu des acides amines ne puisse etre accompli artificiellement. Nous avons entrepris d'installer des deviations systematiques du code genetique chez E. coli dans le but de: (i) produire des proteines aux proprietes chimiques et structurales diversifiees, (ii) relancer l'evolution de souches microbiennes en milieu controle. Ainsi, j'ai construit un mutant defectif de la thymidylate synthase d'E. coli suppressible par incorporation statistique d'azaleucine. Ce mutant a ete obtenu par mutagenese dirigee du gene thyA surexprime a partir d'un plasmide, en substituant un codon Arg par un codon Leu au site 126. D'autres mutants au site 126 s'averent suppressibles par incorporation d'un analogue de la proline, l'azetidine carboxylate. Ces souches, qui montrent une auxotrophie pour un composant metabolique additionnel, procurent des modeles experimentaux pour reconstituer certaines etapes de l'evolution du code genetique. La mutagenese d'un deuxieme exemplaire de la leucyl-tRNA synthetase d'E. coli n'a pas permis d'isoler des variants enzymatiques dont la specificite serait deviee vers l'azaleucine ou d'autres termes de la serie homologue de la methylaminoalanine. J'ai par consequent opte pour la mise en place d'une

enclave traductionnelle chez E. coli, et dans ce but construit des genes
derives de leuX d'E. coli et de cysT de B subtilis exprimant des tRNA SUP L SUP e SUP u et tRNA SUP C SUP y SUP s refractaires a la charge par
les aminoacyl-tRNA synthetases cellulaires, et susceptibles d'etre recrutes
pour former des aminoacyl-tRNA non-canoniques. Enfin, j'ai construit a
partir d'E. coli un prototype genetique compo

17/7/65 (Item 5 from file: 144)
DIALOG(R)File 144:Pascal

09564726 PASCAL No.: 91-0355156
Tempeature sensitivity caused by missense suppressor supH and amber suppressore supP in Escherichia coli
THORBJARNARDOTTIR S; BJOERNSSON A; AMUNDADOTTIR L; EGGERTSSON G
Univ. hosp., inst. biology, Reykjavik 108, Ireland
Journal: Journal of bacteriology, 1991, 173 (1) 412-416
ISSN: 0021-9193 CODEN: JOBAAY Availability: INIST-2041; 354000019765480570/NUM; INIST; 354000019765480570/NUM
No. of Refs.: 26 ref.
Document Type: P (Serial) ; A (Analytic)
Country of Publication: USA
Language: English

17/7/66 (Item 6 from file: 144)
DIALOG(R)File 144:Pascal

00994604 PASCAL No.: 76-0185608
MUTATIONAL PROPERTIES OF SUP P AMBER-OCHRE SUPERSUPPRESSORS IN SACCHAROMYCES CEREVISIAE
GERLACH W L
DEP. GENET., UNIV. ADELAIDE,AUSTRALIA
Journal: MOLEC. GEN. GENET., 1976, 144 (2) 213-215
Availability: CNRS-3571
No. of Refs.: 12 REF.
Document Type: P (SERIAL); DU (DUPLICATION) ; A (ANALYTIC)
Country of Publication: FEDERAL REPUBLIC OF GERMANY
Language: ENGLISH
LES RESULTATS SONT COMPATIBLES AVEC LA PROPOSITION QUE LE LOCUS
SUPP CODE POUR UNE PROTEINE

17/7/67 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

16441665 PMID: 15961494

Automatic detection of subsystem/pathway variants in genome analysis.

Ye Yuzhen; Osterman Andrei; Overbeek Ross; Godzik Adam
Program in Bioinformatics and Systems Biology, The Burnham Institute
10901 N. Torrey Pines Road, La Jolla CA 92037, USA. yye@burnham.org

Bioinformatics (Oxford, England) (England) Jun 2005, 21 Suppl 1
pi478-86, ISSN 1367-4803--Print Journal Code: 9808944
Contract/Grant No.: U54 RR020843; RR; United States NCRR
Publishing Model Print
Document type: Journal Article; Research Support, N.I.H., Extramural
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

MOTIVATION: Proteins work together in pathways and networks, collectively comprising the cellular machinery. A subsystem (a generalization of pathway concept) is a group of related functional roles (such as enzymes) jointly involved in a specific aspect of the cellular machinery. Subsystems provide a natural framework for comparative genome analysis and functional annotation. A subsystem may be implemented in a number of different functional variants in individual species. In order to reliably project functional assignments across multiple genomes, we have to be able to identify the variants implemented in each genome. The analysis of such variants across diverse species is an interesting problem by itself and may provide new evolutionary insights. However, no computational techniques are presently available for an automated detection and analysis of subsystem variants. RESULTS: Here we formulate the subsystem variant detection problem as finding the minimum number of subgraphs of a subsystem, which is represented as a graph, and solve the optimization problem by integer programming approach. The performance of our method was tested on subsystems encoded in the SEED, a genomic integration platform developed by the Fellowship for Interpretation of Genomes as a component of a large-scale effort on comparative analysis and annotation of multiple diverse genomes. Here we illustrate the results obtained for two

expert-encoded subsystems of the biosynthesis of Coenzyme A and FMN/FAD
cofactors. Applications of variant detection, to support genomic annotations and to assess divergence of species, are briefly discussed in
the context of these universally conserved and essential metabolic
subsystems. SUPPLEMENTARY INFORMATION: The details of the variant detection results are available at http://ffas.burnham.org/svar/supp.html.
Record Date Created: 20050617
Record Date Completed: 20060622

17/7/68 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

15055286 PMID: 12584124
GenePath: a system for automated construction of genetic networks from
mutant data.
Zupan Blaz; Demsar Janez; Bratko Ivan; Juvan Peter; Halter John A; Kuspa
Adam; Shaulsky Gad
University of Ljubljana, Faculty of Computer and Information Science
Jozef Stefan Institute, Ljubljana, Slovenia.
Bioinformatics (Oxford, England) (England) Feb 12 2003, 19 (3)
p383-9, ISSN 1367-4803--Print Journal Code: 9808944
Contract/Grant No.: P01 HD39691-01; HD; United States NICHD
Publishing Model Print
Document type: Evaluation Studies; Journal Article; Research Support,
Non-U.S. Gov't; Research Support, U.S. Gov't, Non-P.H.S.; Research Support,
U.S. Gov't, P.H.S.
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
MOTIVATION: Genetic networks are often used in the analysis of biological
phenomena. In classical genetics, they are constructed manually from
experimental data on mutants. The field lacks formalism to guide such
analysis, and accounting for all the data becomes complicated when large
amounts of data are considered. RESULTS: We have developed GenePath, an
intelligent assistant that automates the analysis of genetic data. GenePath

employs expert-defined patterns to uncover gene relations from the data,
and uses these relations as constraints in the search for a plausible
genetic network. GenePath formalizes genetic data analysis, facilitates the
consideration of all the available data in a consistent manner, and the
examination of the large number of possible consequences of planned
experiments. It also provides an explanation mechanism that traces every
finding to the pertinent data. AVAILABILITY: GenePath can be accessed at
http://genepath.org. SUPPLEMENTARY INFORMATION: Supplementary material is
available at http://genepath.org/bi-.supp
  Record Date Created: 20030213
  Record Date Completed: 20030606

 17/7/69 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07356292 PMID: 6745359 Record Identifier: 84261792
  Purkinje cell activity in the primate flocculus during optokinetic
stimulation, smooth pursuit eye movements and VOR-suppression.
  Buttner U; Waespe W
  Experimental brain research. Experimentelle Hirnforschung.
Experimentation cerebrale (GERMANY, WEST) 1984, 55 (1) p97-104,
ISSN 0014-4819--Print Journal Code: 0043312
  Publishing Model Print
  Document type: Journal Article; Research Support, Non-U.S. Gov't
  Languages: ENGLISH
  Main Citation Owner: NLM
  Other Citation Owner: NASA
  Record type: MEDLINE; Completed
  Purkinje cell (PC) activity in the flocculus of trained monkeys was
recorded during: 1) Vestibular stimulation in darkness. 2) Suppression of
the vestibulo-ocular reflex (VOR-supp) by fixation of a small light spot stationary with respect to the monkey. 3) Visual-vestibular conflict
(i.e. the visual surround moves together with the monkey during vestibular
stimulation), which leads to attenuation or suppression of vestibular
nystagmus. 4) Smooth pursuit eye movements. 5) Optokinetic nystagmus
(OKN).

6) Suppression of nystagmus during optokinetic stimulation (OKN-supp) by fixation of a small light spot; whereby stimulus velocity corresponds then to image slip velocity. Results were obtained from PCs, which were activated with VOR-supp during rotation to the ipsilateral side. The same PCs were also modulated during smooth pursuit and visual-vestibular conflict. No tonic modulation during constant velocity OKN occurred with slow-phase nystagmus velocities below 40-60 deg/s. Tonic responses were only seen at higher nystagmus velocities. Transient activity changes appeared at the beginning and end of optokinetic stimulation. PCs were not modulated by image slip velocity during OKN-supp. The results show that in primates the same population of floccular PCs is involved in different mechanisms of visual-vestibular interaction and that smooth pursuit and certain components of OKN slow-phase velocity share the same neural pathway. It is argued that the activity of these neurons can neither be related strictly to gaze, eye or image slip velocity; instead, their activity pattern can be best interpreted by assuming a modulation, which is complementary to that of central vestibular neurons of the vestibular nuclei, in the control of slow eye movements.
  Record Date Created: 19840831
  Record Date Completed: 19840831

17/7/70 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

06090127 PMID: 45219
  Regulation of biosynthesis of aminoacyl-transfer RNA synthetases and of transfer-RNA in Escherichia coli.
  Morgan S; Larossa R; Cheung A; Low B; Soll D
  Archivos de biologia y medicina experimentales (CHILE) Oct 1979, 12 (3) p415-26, ISSN 0004-0533--Print Journal Code: 0321546
  Contract/Grant No.: CA06519; CA; United States NCI; GM22854; GM; United States NIGMS; HD09167; HD; United States NICHD
  Publishing Model Print

Document type: Journal Article; Research Support, U.S. Gov't, P.H.S.
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
We have isolated temperature resistant revertants from temperature sensitive E. coli strains containing either a thermolabile glutaminyl-tRNA synthetase or leucyl-tRNA synthetase. Among the revertants which still contained the thermolabile leucyl-tRNA synthetase we found two classes of regulatory mutants (leuX and leu Y) which have elevated levels of this enzyme. The leuX mutation specifies an operator-promoter region adjacent to the structural gene (leuS) for the enzyme. The leuY gene maps away from the leuS gene and codes for a protein. Using these mutants we demonstrated that the levels of leucyl-tRNA are related to the derepression of the leucine and isoleucine-valine operons. Among the revertants which still contained the thermolabile glutaminyl-tRNA synthetase were characterized three classes of mutants, glnT, glnU, and glnR. The glnT and glnU mutants contain elevated levels of tRNAgln, while the glnR mutant possesses elevated levels of glutaminyl-tRNA synthetase.
The level of glutamine synthetase, the enzyme responsible for the formation of glutamine, is also derepressed in the glnT and glnR mutants.
Record Date Created: 19810116
Record Date Completed: 19810116

17/7/71 (Item 1 from file: 156)
DIALOG(R)File 156:ToxFile

1066246 NLM Doc No: CRISP/2000/ES21227-04 Sec. Source ID:
CRISP/2000/ES21227-04
RELEVANCE OF CHEMICALLY INDUCED HEMANGIOSARCOMAS IN B6C3F1 MOUSE
SILLS RC
NIEHS, NIH
Source: Crisp Data Base National Institutes of Health
Pub. Year: 2000
Sponsoring Agency: U.S. DEPT. OF HEALTH AND HUMAN SERVICES; PUBLIC HEALTH SERVICE; NATIONAL INSTITUTES OF HEALTH, NATIONAL INSTITUTE OF ENVIRONMENTAL

HEALTH SCIENCES
Award Type: Intramural Project
Document type: Research
Languages: ENGLISH
Record type: Completed
Summary of Work: It is hypothesized that epoxide intermediates play a role in t he pathogenesis of hemangiosarcomas by causing genetic alterations in tumor supp ressor genes and proto-oncogenes. Preliminary data has shown that chloroprene i nduced hemangiosarcomas have elevated p53 protein and specific point mutations h ave been detected in exon 5-8. Chemically induced hemangiosarcomas in 1, 3 butad iene o-nitrotoluene and tetrafluoroethylene studies are being evaluated for spec ific genetic alterations in the P53 gene. Tumor suppressor gene and oncogene a nalysis will provide valuable information on the relationship between carcinogen exposure and DNA damage and the relevance to humans.
Record Date Created: 200108

17/7/72 (Item 2 from file: 156)
DIALOG(R)File 156:ToxFile

1064969 NLM Doc No: CRISP/2000/DK56105-02 Sec. Source ID:
CRISP/2000/DK56105-02
FOOD FOLATE FORTIFICATION EFFECT ON FOLATE STATUS
SELHUB J
TUFTS UNIVERSITY, 711 WASHINGTON ST, BOSTON, MA 02111
Source: Crisp Data Base National Institutes of Health
City or State: MASSACHUSETTS Zip Code: 02111
Pub. Year: 2000
Sponsoring Agency: U.S. DEPT. OF HEALTH AND HUMAN SERVICES; PUBLIC HEALTH SERVICE; NATIONAL INSTITUTES OF HEALTH, NAT INST OF DIABETES AND DIGESTIVE AND KIDNEY DISEASES
Award Type: Grant
Document type: Research
Languages: ENGLISH
Record type: Completed
In 1996, the U.S. Food and Drug Administration published a regulation to be effe ctive by January 1998 that all enriched fluor, breads, rive, pasta,

corn meal, a nd other grain products would contain 140 micrograms of folic acid per 100 grams . The main purpose of this proposed study is to examine the impact of fortificat ion on folate status in the Framingham Offspring Study cohort. We have measured plasma folate and homocysteine concentrations at the 5th examination cycle (Jan uary 1991-December 1994), and have preliminary data on a subset of subjects from the 6th examination cycle (January 1995-September 1998). We propose to measure folate and homocysteine status at the 7th examination cycle (September 1998-Augu st 2001) so that we can establish post-fortification concentrations of plasma fo late and homocysteine in the entire cohort. We plan to test the following hypoth eses: 1) Current folic acid fortification levels are adequate to virtually elimi nate low folate status and elevated homocysteine concentrations associated with inadequate folate status in relatively healthy, non- institutionalized adults. 2) Folic acid fortification at current levels will largely remove the methylenete trahydrofolate reductase (MTHFR) C677T mutation as a risk factor for elevated ho mocysteine concentrations. 3) The increase in folate intake due to fortification will increase the prevalence of low vitamin B12 status in association with fola te intakes above 1 microgram/day. 4) The important sources of folate in the diet are dramatically changed in the area of folic acid fortification. The following specific aims address each of these hypotheses in the Framingham Offspring coho rt, a population-based sample of adults, in which three serial blood samples wil l be obtained between the years 1991 and 2001 (with at least one sample obtained before and after implementation of fortification) in combination with members o f the Framingham Omni minority cohort, which was established between 1994 and 19 98: 1) To assess the change in plasma and RBC folate and plasma

homocysteine con centrations associated with the FDA-RBC folate at the 7th examination cycle, and RBC folate at the 6th examination. 2) To determine if the relation between homo cysteine and MTHFR C677T genotype is weakened after implementation of folic acid fortification, we will determine MTHFR genotype for the C677T mutation in the O ffspring cohort. 3) To evaluate the potential for an increased prevalence of low vitamin B12 status in the presence of high folic acid intakes after fortificati on, we will measure plasma vitamin B12 and dietary supplemental nutrient intake using the Willett food frequency questionnaire at examination 7. 4) To determine the contribution of folic acid from fortification of folic acid from fortificat ion to total dietary folate and the change in percent contribution for each sour ce before and after fortification so that we can characterize the most important sources of folate in the era of fortification, we will measure dietary and supp lemental nutrient intake using the Willett food frequency questionnaire at exami nation 7.

Record Date Created: 200108

17/7/73 (Item 3 from file: 156)
DIALOG(R)File 156:ToxFile

1058130 NLM Doc No: CRISP/1999/BC05748-07 Sec. Source ID:
CRISP/1999/BC05748-07

GENOMIC ALTERATIONS IN FOOD-DERIVED HETEROCYCLIC AMINE-INDUCED RAT MAMMARY GLAND

SNYDERWINE E
NCI BC, NIH

Source: Crisp Data Base National Institutes of Health
Pub. Year: 1999
Sponsoring Agency: U.S. DEPT. OF HEALTH AND HUMAN SERVICES; PUBLIC HEALTH SERVICE; NATIONAL INSTITUTES OF HEALTH, DIVISION OF BASIC SCIENCES - NCI
Award Type: Intramural Project
Document type: Research
Languages: ENGLISH

Record type: Completed

2-Amino-1-methyl-6-phenylimidazo[4,5-b]pyridine (PhIP), a mutagenic compound fou nd the human diet in cooked meat, is a mammary gland carcinogen in female Spragu e-Dawley rats. PhIP-induced rat mammary gland carcinomas were examined for mutat ions in several genes (exons) known to regulate cell growth and apoptosis includ ing p53 (4-8), p21Waf1 (coding region), Apc (14, 15), B-catenin (3), E-cadherin (9,13,15), Bcl-x (coding region), Bax (3), IGFIIR (28), and TGFBIIR (3). DNA fro m 30 carcinomas were examined by single strand conformation polymorphism analysi s but no mutations were detected in these genes/gene regions. DNA from carcinoma s and matching normal tissue were further screened for allelic imbalance using a polymerase chain reaction-based approach with primers to known microsatellite r egions located throughout the rat genome. Out of 53 markers examined, twelve rev ealed allelic imbalance. Microsatellite instability (MSI) was detected at two ma rkers, one on chromosome 4 and one on chromosome 6. Sixty-five percent and 96% o f all carcinomas examined (N=23) showed MSI at these loci on chromosomes 4 and 6 , respectively, supporting the notion that MSI plays a role in PhIP-induced mamm ary carcinogenesis. Loss of heterozygosity (LOH), an indication of a possible tu mor suppressor gene, was observed at ten markers distributed on chromosomes 3, 1 0, 11, 14, and X. The frequency of LOH at these markers ranged from 75%-94% supp orting that the regions of allelic imbalance were largely similar for the PhIP-i nduced carcinomas examined in this study. When PhIP-induced carcinomas from rats placed on high fat and low fat diet were compared, no unique regions of allelic imbalance nor statistical differences in the frequency of allelic imbalance wer e observed. The results indicate that the high fat diet, known to be a promoter of PhIP-induced rat mammary carcinogenesis, did not influence

allelic imbalance in the carcinomas. Interestingly, DMBA-induced mammary carcinomas did not show a llelic imbalance at 11 of the 12 loci which showed allelic imbalance in PhIP-ind uced carcinomas. These findings suggest that distinct chemical carcinogens induc e different patterns of allelic imbalance during rat mammary carcinogenesis. Fur ther studies are needed to determine whether regions of LOH harbor potentially n ovel tumor suppressor genes involved in breast cancer. - Breast cancer, Allelic imbalance, Heterocyclic amines, Diet, Microsatellite instability, Loss of hetero zygosity, Rat,
Record Date Created: 200010

17/7/74 (Item 4 from file: 156)
DIALOG(R)File 156:ToxFile

174142 NLM Doc No: DART/TER/95001633 Sec. Source ID: DART/TER/95001633
Placental lysyl oxidase RNA expression after vitamin E supplementation and prenatal alcohol exposure in rats.
Puryear TK; Schildkrout A; Krawetz SA; Hannigan JH
Fetal Alcohol Research Center, Wayne State University, Detroit, MI.
Source: Alcohol Clin Exp Res 1994 Apr;18(2):471
Journal Name: Alcohol Clin Exp Res Pub. Year: 1994 ISSN: 0145-6008
Contract/Grant No.: NIAAA06721; NIAAA07531; NIAAA07606
Document type: ABSTRACT
Languages: ENGLISH
Record type: Completed
We studied nutritional deficiency in Vitamin E (VitE) as a potential risk factor for FAS. VitE is an essential nutrient that protects cells from cytotoxic free radicals that are increased by alcohol. We hypothesized that VitE deficiency would exacerbate and VitE supplementation would attenuate alcohol-induced fetopathy in rats. Female rats were fed VitE DEFicient diets (0 IU/kg) for 2 weeks before mating. From gestation day 0 (GD0) dams either ate DEF diet, or diets with NORMal (35 IU/kg) or SUPPlemented (70 IU/kg) levels of VitE. From GD8 to GD20, dams were

intubated with 0, 3 or 5 g/kg/day of ethanol. Controls were unintubated. On GD20, 30 min after intubation, maternal blood, amniotic fluid, fetuses and placentae were collected. EtOH decreased litter size and increased placental weight. Maternal weight gain and fetal weight were reduced additively by VitE DEF and EtOH. VitE DEF decreased fecundity. While EtOH effects were unchanged, VitE DEF decreased and VitE SUPP increased placental lysyl oxidase RNA levels. The results suggest that VitE DEF can exacerbate some signs of FAS (e.g. low fetal weight), but that VitE SUPP may not ameliorate fetal alcohol effects.
Record Date Created: 199601

17/7/75 (Item 1 from file: 369)
DIALOG(R)File 369:New Scientist

00126264 16622434.800 (THIS IS THE FULLTEXT)
Left right and wrong
AINSWORTH, CLAIRE; Claire Ainsworth is a science writer living in London
New Scientist, vol. 166, no. 2243, p. 40
June 17, 2000

TEXT: When an embryo can't tell one side from the other the result can be catastrophic. Claire Ainsworth sorts out the confusion

CHECKED your insides recently? They could be the wrong way round. No kidding. The chances are that at least 75 New Scientist readers are the mirror image of the rest of us, and they're probably none the wiser.

Most of us don't give our internal body plans a second thought. You've probably been taught that your heart lies on the left-hand side of your chest, and if you've got a right-sided pain in your belly you start getting paranoid about your appendix. But for one in 8500 people, the exact opposite is true. It's as though they had stepped through a mirror into looking-glass land: the handedness and placing of their internal organs are completely reversed. It's a secret that many of them carry to the grave,

because the reversal is so perfect they never even notice.

But these people are the lucky ones. An unlucky few find themselves stuck halfway through the mirror in a deadly limbo where their body pattern is neither fully one way nor the other. Their internal organs can get tangled up in a life-threatening jumble, and many need emergency surgery as soon as they are born. Even partial forays into looking-glass land can land you in trouble: minor disruptions of this left-right patterning process could be a leading cause of congenital heart problems in newborn babies.

Curiouser and curiouser indeed, but how do these problems arise? You might think that college-educated biologists would have figured out how to tell left from right by now. But for an embryo it's not that simple. How it does this, and then sets up its internal asymmetry, has puzzled developmental biologists for decades. At last, though, researchers think they may have found the answer in a thoroughly unexpected place: a tiny molecular motor that waves things to the left like a police officer directing traffic.

Packing puzzle

Why should our internal organs be asymmetric in the first place? For two main reasons, says Joseph Yost, a developmental biologist at the University of Utah. The first is a straightforward packing problem--how to fit everything together in a restricted space. The lungs, for example, have to share space in the chest with the heart and its great blood vessels. To make room, your left lung has two lobes and your right has three, and the major airways, the left and right bronchi, are tilted at different angles. The upshot is that if you manage to inhale a peanut in some bizarre drunken accident at a party, it's practically guaranteed to lodge in your right lung. A neat piece of trivia to test the doctors on when you finally stagger, hacking and spluttering, into your local casualty department.

The second reason is to do with making organs function efficiently,

and making sure that they link up to each other properly. Nowhere is this more apparent than in the heart, says Nigel Brown, a heart development expert at St George's Hospital Medical School in London. Researchers in Britain have recently confirmed that the heart's asymmetric design improves blood flow and makes it an extremely efficient pump. In addition, the right side of the heart is smaller than the left, because it collects blood from the body and only has to pump it across to the lungs, while the left side has to pump oxygenated blood all the way round the body. So not only does the heart have to be asymmetric itself, it must also be plumbed into the lungs and the body the correct way.

If your left-right pattern is completely reversed, a condition called situs inversus, there's no problem, because all the organs are reversed with respect to each other (see Diagram). (Curiously, such people have no more chance of being left-handed than the rest of the population.) But between there and normality lies dangerous ground. "Anything in between and you're in various degrees of trouble, depending on exactly what's wrong," says Brown.

CAPTION: How body organs may be reversed (FIGURE OMITTED)

The most extreme problem is called isomerism. In this condition, the organs are entirely symmetrical, as if you're standing with a mirror placed down the midline of your body. The way your organs are affected depends on which side of your body is being reflected in the mirror. If it's the right side, your spleen will be missing and both your lungs will have three lobes. Worst of all, your heart will be symmetrical, too.

Too right

A heart with two right sides is a double whammy of bad news. First, the chambers won't be strong enough to pump blood around the body. And even if they could manage, you run into a second problem, which is that the blood vessels that connect other organs to the heart don't know where to

attach. So they just plug themselves in randomly to the nearest blood vessels, such as the hepatic vein, which carries blood from the liver. It's as if someone's done a cowboy job on the body's plumbing. Babies born with a double-right heart need urgent surgery to have any hope of surviving.

But you don't have to go to anything like this extreme to be affected. A mix of organs, some the right way and others inverted--a condition called heterotaxia--also lands you with plumbing problems. More subtle problems with left-right patterning could affect many of the 8 per 1000 British children born with congenital heart defects, says Brown.

As well as being important clinically, understanding left-right patterning is a challenging intellectual problem that has puzzled developmental biologists for years. When an embryo starts to develop, it needs to find its bearings so that it knows where different parts such as legs and guts should grow. So it establishes reference lines or "axes" that run from head to tail and belly to back, marking which end is the head and which side is the front. Biologists have a fair idea of how the embryo does this, but the left-right decision is trickier. To establish left-right asymmetry, an embryo must break its bilateral symmetry in a consistently handed fashion, and set up a new axis exactly perpendicular to the other two.

So what immortal hand or eye could break bilateral symmetry? In the early 1990s, Brown and Lewis Wolpert, a developmental biologist now at University College London, suggested a mechanism. Imagine a molecule in the shape of the letter F, they said. No matter how hard you try, you can never superimpose this F molecule on a mirror image of itself with the same side facing upward--in other words, it's "chiral". This intrinsic handedness means that embryos could use the F molecule to reliably tell their left hand from their right (see Diagram).

CAPTION: Left-right patterning on a mouse (FIGURE OMITTED)

That's fine and dandy in theory, but finding this hypothetical molecule has been another matter. For a start, nobody knew what it was, or even what it looked like. Biologists have built up a fairly good picture of

the genes and biochemical pathways that act once the symmetry is broken.
But it's only now that the F molecule's identity may have been unmasked.

The first clue came in 1976 from studies on a group of people with a rare condition called Kartagener's syndrome. These people show a range of left-right problems, and intriguingly, the men are infertile. When Bjorn Afzelius of Stockholm University took a closer look he found that the sperm tails, or flagella, of these men are paralysed, as are similar hair-like structures called cilia on other cells. What was the link between cilia and left-right asymmetry? Afzelius suggested a connection. When cilia beat, he said, they might force developing organs to bend in a particular direction.
Nobody paid much attention to this idea at the time, but 22 years later, a chance discovery by researchers in Japan pushed cilia back into the limelight.

Nobutaka Hirokawa and his colleagues at Tokyo University were studying a group of proteins called kinesins. These are the packhorses of the cell, large protein complexes that shoulder packages called vesicles that contain anything from chemical messengers that cells use to talk to each other to the bricks and mortar needed to build cellular structures.
Kinesins tramp up or down the cell's scaffolding, the cytoskeleton, to deliver their load to its required destination. Hirokawa's group had identified a new kinesin complex in mice, and to find out exactly what it was doing they genetically engineered mice that lacked part of the complex.

All these mice died as young embryos --and about half turned out to have reversed left-right patterning. Intrigued by this result, the researchers took a closer look at very young embryos, specifically at a structure called the node that was known to be important for dictating left-right asymmetry. The node is a triangular patch of cells that forms a pit at the head end of the developing embryo before marching down the embryo laying down cells that establish the head-tail axis. It's the embryo's Mission Control, telling cells where to go and what to do. The node is normally covered in cilia, but those of Hirokawa's mice were bare.

Without the special kinesin, the cells couldn't build any.

However, this still didn't explain why half the mice had situs inversus. Studying the nodal cilia under an electron microscope only deepened the mystery, as they turned out to have an unusual structure. Cilia are usually made up of nine pairs or "doublets" of tubes called microtubules arrayed around two central singlets: this is the classical "9+2" arrangement. Large protein motors called dyneins form arms which link the outer tubule of one doublet with the inner tubule of the next. The action of these motors forces the doublets to slide past each other lengthways, driving the lashing motion of the cilia in the process, although nobody knows exactly how.

The nodal cilia have a slightly different structure that lacks the central pair, and scientists have generally assumed that these sorts of structures, called monocilia, don't beat. Biologists have largely thought of them as sensory structures: the light-detecting rods and cones in our retinas, for example, are monocilia. Just to be sure, though, Hirokawa's group watched these cilia in normal living mouse embryos, using a sophisticated wide-angled microscope to provide maximum depth of field. To their astonishment, they found that the cilia were moving. But instead of following the whip-like motion of 9+2 cilia, they were whizzing around clockwise like tiny propellers. "This was a very big surprise," says Hirokawa. When the researchers added tiny fluorescent beads to the fluid around the embryos, the beads were consistently wafted from right to left. Suddenly, the penny dropped. What if, instead of beads, this "nodal flow" wafted a chemical signal over to mark the left side of the embryo?

It's an intriguing possibility, but Hirokawa's mutant mice alone don't prove that cilia hold the smoking gun. After all, kinesins could influence a number of cellular processes, and it's not just asymmetry that's affected in the mutant embryos. So how do we know that nodal cilia are anything more than gyrating red herrings?

Well, we don't know for sure yet. Nodal cilia are tiny, and hardly anyone has actually witnessed them moving because they are so hard to see, so many researchers remain sceptical. One of the doubters was Martina Brueckner, a cardiologist at Yale University, who wanted to see the cilia for herself. "I spent most of last winter struggling with this using every

microscope on this campus," she recalls. "I couldn't see it." Finally, she managed to borrow a microscope with a very wide-angle lens like Hirokawa's--and there the cilia were, twirling away. "They're there," she says now. "It's real."

Brueckner had a special interest in the outcome because she had been studying mice with a mutated gene called inversus viscerum, or iv, which causes various forms of left-right reversal similar to those seen in people. When she and her colleagues isolated the gene, it turned out to encode a dynein--they called it left-right dynein--that was similar to the dyneins found in the outer arms of cilia. The team then genetically engineered a mouse strain that lacked only the head-end of the dynein, which is crucial for its motor function. Under the borrowed microscope, the nodal cilia on her engineered mice stood stock-still like a battalion of tin soldiers, their dynein motors jammed. And sure enough, the mice had situs inversus. "This supports our hypothesis very well," says Hirokawa. The case for nodal flow was starting to look stronger.

So are cilia truly the movers and shakers of symmetry breaking? To find out when the cilia and nodal flow were active, Hirokawa and his team looked at the expression pattern of a gene involved in left-right asymmetry. It is the earliest-acting gene known so far, and is normally expressed only on the left-hand side of the developing embryo. In the mutant mice, however, it was expressed either on both sides or not at all. So whatever process is going wrong in Hirokawa's mutants, it acts earlier than any other known step, possibly forming the initial symmetry-breaking mechanism that biologists are searching for.

Moreover, cilia themselves have all the qualities that researchers were looking for in the elusive F molecule. Imagine you've clambered up a nodal cell's cytoskeleton, and are peering up a cilium as it towers out of the cell--a beautifully intricate molecular machine. Look at the way it's constructed, and you'll notice that the cilium itself is asymmetric (see Diagram). The dynein arms that link the nine outer doublets are all angled

to the right, like spokes on a pinwheel. Cut a slice across the cilium, and you will find that it cannot be superimposed on a mirror-image of itself: the structure is chiral. Could this whole massive structure fulfil the role of the handed molecule postulated by Brown and Wolpert? "If you broaden the concept at that level," says Brueckner, "then the cilium is a perfectly beautiful F molecule." Combined with the shape of the node, the clockwise twirling of the cilia generates the leftward flow.

CAPTION: The asymmetric pattern of the cilium (FIGURE OMITTED)

It's an elegant solution to a complex problem, but the jury is still out, and the response to Hirokawa's nodal flow model has been mixed. Most developmental biologists are intrigued, but cautious. Intellectually, it's a very appealing model, says Brown, but he questions whether the subtle wafting currents would be robust enough to do the trick reliably. Another problem with the theory is that researchers have yet to pinpoint an equivalent population of cilia in other species. Finding them in other animals--especially in standard lab creatures such as chicks and frogs--would add more weight to the idea, says Yost. "My general sense is that the cilia [model] is a very exciting possibility, but there are still a few issues that need to be resolved--as far as knowing that they are the instigators of left-right patterning," he adds.

Wolpert is also hesitant to hail the unmasking of his elusive F molecule just yet. "I think it's a remarkable observation, but you've got to be a little careful," he says. "How does one know that it is the cilia that are doing it, and not just a mutation that affects something else and the cilia?" His reservations are echoed by Denys Wheatley, a cell biologist at the University of Aberdeen. We need more robust evidence that the twirling motion of cilia actually causes left-right patterning, he says.

What would convince the doubting Thomases? If someone were to use a jet of water to interfere with the nodal flow, says Wolpert, then he'd be impressed. "It's a trivial experiment," he laments. "I can't understand why

they don't do it." But Kyle Vogan, a researcher at Harvard Medical School who studies chick development, is enthusiastic about the theory. "It does have the power to explain some of the most challenging issues related to left-right patterning," he says. The problem of finding similar cilia in other animals could all be down to timing, he says. They may be active at different stages in different species.

If Hirokawa's theory turns out to be right, it opens up a whole new understanding of how embryos tell left from right and how this critical asymmetry goes wrong. The irony is that the human race has always knocked asymmetry, seeing it as a sign of imperfection. Your symmetrical outside might make you a beauty, but it's your asymmetrical insides that keep you alive. It's something to ponder as you inspect yourself in the mirror in the morning. The next time you claim your heart's in the right place, you may want to think again.

References and Notes:

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00500056 (USE 9 FOR FULLTEXT)
Determining Divergence Times of the Major Kingdoms of Living Organisms with a Protein Clock
Doolittle, Russell F.; Feng, Da-Fei; Tsang, Simon; Cho, Glen; Little, Elizabeth
The authors are at the Center for Molecular Genetics University of California, San Diego, La Jolla, CA 92093-0634, USA.
Science Vol. 271 5248 pp. 470
Publication Date: 1-26-1996 (960126) Publication Year: 1996
Document Type: Journal ISSN: 0036-8075
Language: English

Section Heading: Articles
Word Count: 5901

Abstract: Amino acid sequence data from 57 different enzymes were used
to determine the divergence times of the major biological groupings.
Deuterostomes and protostomes split about 670 million years ago and plants,
animals, and fungi last shared a common ancestor about a billion years ago.
With regard to these protein sequences, plants are slightly more similar to
animals than are the fungi. In contrast, phylogenetic analysis of the same
sequences indicates that fungi and animals shared a common ancestor more
recently than either did with plants, the greater difference resulting from
the fungal lineage changing faster than the animal and plant lines over the
last 965 million years. The major protist lineages have been changing at a
somewhat faster rate than other eukaryotes and split off about 1230 million
years ago. If the rate of change has been approximately constant, then
prokaryotes and eukaryotes last shared a common ancestor about 2 billion
years ago, archaebacterial sequences being measurably more similar to
eukaryotic ones than are eubacterial ones.

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with regard to organismic representation. Sequences for enzymes encoded by organellar DNA (mitochondria and chloroplasts) and sequences from viruses were not included. The sequences of candidate groups were aligned and phylogenies were constructed (B17) (B18) (B19) (B20) (B21) (B22) . If the phylogenetic trees seemed reasonable, by which we mean there was no evidence of horizontal gene transfer or adulteration by paralogous comparisons (B23) , the sequence subset became a part of the study. The entire set (divided into the six standard enzyme groups) can be obtained by anonymous ftp from juno.ucsd.edu. cd to directory pickup. ;

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As new data become available, it is relatively easy to update the records and recalculate all values. ;

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23. Several situations in which displacement during evolution has led to functional convergence after paralogous radiations include bacterial ornithine decarboxylase (E.C. 4.1.1.17), which is obviously more like lysine decarboxylase (E.C. 4.1.1.18) than it is to eukaryotic ornithine decarboxylases, and bacterial tyrosine transaminase (E.C. 2.6.1.5), which is more similar to bacterial aspartate transminase (E.C. 2.6.1.1) than it is to the eukaryotic tyrosine enzyme. Other enzyme sets that were not included on the basis of anomalous phylogenetic trees were catalase (E.C. 1.11.16), pyrroline carboxylate reductase (E.C. 1.5.1.2), glutathione reductase (E.C. 1.6.4.2), phosphoribosylglycineamide formyl transferase (E.C. 2.1.2.2), transketolase (E.C. 2.2.1.1), glycogen phosphorylase (E.C. 2.4.1.1), hypoxanthine transferase (E.C. 2.4.2.8), orotate phosphate transferase (E.C. 2.4.2.10), glutathione transferase (E.C. 2.5.1.18), galactokinase (E.C. 2.7.1.6), adenylate kinase (E.C. 2.7.4.3), uridine 5 (prime) -diphosphate-glucose-hexose phosphate uridyl transferase (E.C. 2.7.7.9 and E.C. 2.7.7.12), ornithine decarboxylase (E.C. 4.1.1.17), glucose phosphate isomerase (E.C. 5.3.1.9), and phosphoglycerate mutase (E.C. 5.4.2.1). ;
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25. Average percent identities notwithstanding, the data were not entirely consistent. Of the nine possible comparisons, in five cases the archaebacterial sequences clustered with the eukaryotes, and in three with the eubacteria. In one case (phosphoglycerate kinase, E.C. 2.7.2.3)

the eubacteria and eukaryote sequences were more similar to each other than to the archaebacterial sequence. ;
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Proc. Natl. Acad. Sci., U.S.A., 86 1989, 9355 ;
44. Ochman, H., Wilson, A. C., J. Mol. Evol., 26 1987, 74 ;
45. Matrices of pairwise distances were examined by the program BLEN (B18)
which uses a least squares approach (B46) . ;
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W. M., Langley, C. H., Fed. Proc., 35 1976, 2092 Fitch, W. M.,
Ayala, F. J., Proc. Natl. Acad. Sci. U.S.A., 91 1994, 6802 ;
48. An extreme but valid example is the case of enzyme sequences in
retroviruses. For example, a survey of 26 ribonuclease H sequences
revealed that only 4 of 120 residues remained unchanged; R. F. Doolittle,
D-F. Feng, M. S. Johnson, M. A. McClure, Q. Rev. Biol. 64, 1 (1989). ;
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Press, New York, , ;
50. Although, on the average, eukaryote and eubacteria sequences are 37
percent identical, the observed fraction of irreplaceable residues was,
again on average, only 17 percent. There was also a natural tendency for
the fraction of irreplaceable residues to be smaller; the larger the
number of sequences in a set, the extrapolated fraction being about 5
percent. ;
51. A computer model has been constructed that follows the divergence of
mutated protein sequences under various circumstances of constraint
(R. F. Doolittle and D. F. Feng, in preparation). ;
52. One of the earliest estimates made about the prokaryote-eukaryote
divergence concluded, on the basis of a relatively small number of
transfer RNA sequences, that the split occurred about twice as long ago
as the divergence of plants, animals, and fungi (B6) . ;

53. There will be some who will remind us of alternative scenarios concerning the origin of eukaryotic organisms, and especially of the possibility that some of the sequences discussed here were actually imported by an archaebacterial symbiont destined to become the nucleus.
The fusion of a eubacterial "prokaryote" and an archaebacterium has been
widely discussed (B54) . Although we are skeptical of such models on other grounds, we should point out that such an occurrence would not affect our findings, except that the time we are reporting as a divergence time for eukaryotes and eubacteria would instead chronicle the
alleged fusion event. ;
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Golding, G. B., Gupta, R. S., Mol. Biol. Evol., 12 1995, 1 ;
55. We thank K. Anderson for assistance in preparing this manuscript and
S. Frank, J. Gillespie, and two anonymous reviewers for helpful suggestions. Supported in part by NIH grant HL-26873.

17/7/77 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

144126215 CA: 144(8)126215j JOURNAL
A novel interstitial deletion on the long arm of chromosome 16 in a patient with chronic myelomonocytic leukemia
AUTHOR(S): Kozon, Lukasz K.; Wesley, Deborah L.; Van Brunt, John; Li,
Marilyn M.
LOCATION: Human Genetics Program, Hayward Genetics Center, Tulane University Health Science Center, New Orleans, LA, 70112, USA
JOURNAL: Cancer Genet. Cytogenet. (Cancer Genetics and Cytogenetics)
DATE: 2005 VOLUME: 162 NUMBER: 1 PAGES: 92-94 CODEN: CGCYDF ISSN:
0165-4608 PUBLISHER ITEM IDENTIFIER: 0165-4608(05)00091-9 LANGUAGE: English PUBLISHER: Elsevier Inc.
SECTION:
CA214001 Mammalian Pathological Biochemistry
CA203XXX Biochemical Genetics
IDENTIFIERS: chromosome 16 interstitial deletion myelomonocytic leukemia
leukemogenesis
DESCRIPTORS:
Mutation...
deletion; long arm of chromosome 16 showed interstitial deletion, extending from 16q11.2 to q24 in chronic myelomonocytic leukemia patient suggesting this may results in deletion of tumor suppressor g
Chromosome...
human 16; long arm of chromosome 16 showed interstitial deletion,

extending from 16q11.2 to q24 in chronic myelomonocytic leukemia
patient suggesting this may results in deletion of tumor
suppressor g
Chronic myelomonocytic leukemia... Human...
long arm of chromosome 16 showed interstitial deletion, extending
from
16q11.2 to q24 in chronic myelomonocytic leukemia patient
suggesting
this may results in deletion of tumor suppressor gene and ma
Gene,animal...
tumor suppressor; long arm of chromosome 16 showed interstitial
deletion, extending from 16q11.2 to q24 in chronic myelomonocytic
leukemia patient suggesting this may results in deletion of tumor
supp

17/7/78 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

144125949 CA: 144(8)125949q JOURNAL
Krebs cycle enzymes as tumor suppressors
AUTHOR(S): Baysal, Bora E.
LOCATION: Department of Obstetrics, Gynecology and Reproductive
Sciences,
Otolaryngology and Human Genetics, Magee-Womens Research Institute,
University of Pittsburgh School of Medicine, Pittsburgh, PA, 15213,
USA
JOURNAL: Drug Discovery Today: Dis. Mech. (Drug Discovery Today:
Disease
Mechanisms) DATE: 2005 VOLUME: 2 NUMBER: 2 PAGES: 247-254 CODEN:
DDTDAO UNIFORM RESOURCE LOCATOR (URL):
http://www.sciencedirect.com/science/journal/17406765 MEDIA TYPE:
online
computer file ISSN: 1740-6765 LANGUAGE: English PUBLISHER:
Elsevier B.V.
SECTION:
CA214000 Mammalian Pathological Biochemistry
IDENTIFIERS: review Krebs cycle tumor suppressor succinate
dehydrogenase
fumarate hydratase, paraganglioma hereditary leiomyomatosis renal
cell
cancer syndrome review
DESCRIPTORS:
Gene,animal...
Fumarate hydratase; germline inactivation mutations in Krebs cycle
enzyme fumarate hydratase genes causes hereditary
leiomyomatosis/renal
cell cancer syndrome in human suggesting these as tumor suppre
Kidney...
germline inactivation mutations in Krebs cycle enzyme fumarate
hydratase genes causes hereditary leiomyomatosis/renal cell cancer

syndrome in human suggesting these as tumor suppressors and can be the
Tricarboxylic acid cycle... Human...
mutations in Krebs cycle enzymes succinate dehydrogenase and fumarate
hydratase genes causes paraganglioma and hereditary
leiomyomatosis/renal cell cancer syndrome in human suggesting these as
tumor s
Kidney,neoplasm...
renal cell carcinoma; germline inactivation mutations in Krebs cycle
enzyme fumarate hydratase genes causes hereditary leiomyomatosis/renal
cell cancer syndrome in human suggesting these as tumor supp
Carcinoma...
renal cell; germline inactivation mutations in Krebs cycle enzyme
fumarate hydratase genes causes hereditary leiomyomatosis/renal cell
cancer syndrome in human suggesting these as tumor suppressors an
Gene,animal...
Succinate dehydrogenase; germline inactivation mutations in Krebs cycle
enzyme succinate dehydrogenase genes causes paraganglioma in human
suggesting these as tumor suppressors and can be therapeutic
Gene,animal...
tumor suppressor; germline inactivation mutations in Krebs cycle
enzymes like succinate dehydrogenase and fumarate hydratase genes
causes paraganglioma and hereditary leiomyomatosis/renal cell cancer
CAS REGISTRY NUMBERS:
9032-88-6 germline inactivation mutations in Krebs cycle enzyme fumarate
hydratase genes causes hereditary leiomyomatosis/renal cell cancer
syndrome in human suggesting these as tumor suppressors and can be
therapeutic target
9002-02-2 germline inactivation mutations in Krebs cycle enzyme succinate
dehydrogenase genes causes paraganglioma in human suggesting these as
tumor suppressors and can be therapeutic target

17/7/79 (Item 3 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

143109403 CA: 143(7)109403f JOURNAL
Geranylgeranylacetone ameliorates ischemic acute renal failure via induction of Hsp70
AUTHOR(S): Suzuki, Satoshi; Maruyama, Shoichi; Sato, Waichi; Morita, Yoshiki; Sato, Fumihiko; Miki, Yusuke; Kato, Sawako; Katsuno, Masahisa;

Sobue, Gen; Yuzawa, Yukio; Matsuo, Seiichi
  LOCATION: Division of Clinical Immunology, Department of Medicine, Nagoya
University Graduate School of Medicine, Nagoya, Aichi, Japan,
  JOURNAL: Kidney Int. (Kidney International) DATE: 2005 VOLUME: 67
  NUMBER: 6 PAGES: 2210-2220 CODEN: KDYIA5 ISSN: 0085-2538
LANGUAGE:
English PUBLISHER: Blackwell Publishing, Inc.
  SECTION:
    CA201008 Pharmacology
  IDENTIFIERS: heat shock protein geranylgeranylacetone kidney ischemia
    reperfusion injury renoprotectant
  DESCRIPTORS:
Antiulcer agents...
    antiulcer agent GGA induced Hsp70 and protected tubular epithelial cell
    from apoptosis, inturn ameliorated tubular damage by I/R injury in rat,
    induced Hsp70 and suppressed apoptosis in rat tubular ep
Kidney,disease...
    failure, acute; geranylgeranylacetone induced Hsp70, attenuated tubular
    damage and macrophage infiltration and protected tubular epithelial
    cell from apoptosis, inturn ameliorated ischemic acute renal
Kidney...
    geranylgeranylacetone induced Hsp70, attenuated tubular damage and
    macrophage infiltration and protected tubular epithelial cell from
    apoptosis, inturn ameliorated ischemic acute renal failure in rat
Oxidative stress,biological...
    geranylgeranylacetone induced Hsp70, protected tubular epithelial cell
    from apoptosis, inturn ameliorated tubular damage by I/R injury in rat
    and induced Hsp70 rat tubular epithelial cell culture
Apoptosis...
    geranylgeranylacetone protected tubular epithelial cell from apoptosis,
    inturn ameliorated tubular damage by I/R injury in rat and suppressed
    apoptosis in rat tubular epithelial cell culture
Heat-shock proteins...
    GGA induced Hsp70 but not Hsp90, Hsc70, Hsp60, Hsp32 and protected
    tubular epithelial cell from apoptosis, inturn ameliorated tubular
    damage by I/R injury in rat, induced Hsp70 and suppressed apoptosi
Proteins...
    hsc 70 (heat-shock cognate, 70 kDa); geranylgeranylacetone induced
    Hsp70, protected tubular epithelial cell from apoptosis, inturn
    ameliorated tubular damage by I/R injury in rat and induced Hsp70 rat
Heat-shock proteins...

HSP 32; geranylgeranylacetone induced Hsp70, protected tubular epithelial cell from apoptosis, inturn ameliorated tubular damage by I/R injury in rat and induced Hsp70 rat tubular epithelial cell cult

Heat-shock proteins...

HSP 60; geranylgeranylacetone induced Hsp70, protected tubular epithelial cell from apoptosis, inturn ameliorated tubular damage by I/R injury in rat and induced Hsp70 rat tubular epithelial cell cult

Heat-shock proteins...

HSP 70; geranylgeranylacetone induced Hsp70, protected tubular epithelial cell from apoptosis, inturn ameliorated tubular damage by I/R injury in rat and induced Hsp70 rat tubular epithelial cell cult

Heat-shock proteins...

HSP 90; geranylgeranylacetone induced Hsp70, protected tubular epithelial cell from apoptosis, inturn ameliorated tubular damage by I/R injury in rat and induced Hsp70 rat tubular epithelial cell cult

Reperfusion...

injury; geranylgeranylacetone induced Hsp70, attenuated tubular damage and macrophage infiltration and protected tubular epithelial cell from apoptosis, inturn ameliorated ischemic acute renal failure

Kidney,disease...

ischemia; geranylgeranylacetone induced Hsp70, attenuated tubular damage and macrophage infiltration and protected tubular epithelial cell from apoptosis, inturn ameliorated ischemic acute renal failu

Epithelium...

renal tubular; GGA induced Hsp70 but not Hsp90, Hsc70, Hsp60, Hsp32 and protected tubular epithelial cell from apoptosis, inturn ameliorated tubular damage by I/R injury in rat, induced Hsp70 and supp

Ischemia...

renal; geranylgeranylacetone induced Hsp70, attenuated tubular damage and macrophage infiltration and protected tubular epithelial cell from apoptosis, inturn ameliorated ischemic acute renal failure

Cytoprotective agents...

renoprotective; geranylgeranylacetone induced Hsp70, protected tubular epithelial cell from apoptosis, inturn ameliorated tubular damage by I/R injury in rat and induced Hsp70 rat tubular epithelial c

Injury...
reperfusion; geranylgeranylacetone induced Hsp70, attenuated tubular
damage and macrophage infiltration and protected tubular epithelial
cell from apoptosis, inturn ameliorated ischemic acute renal fa
Kidney...
tubule, epithelium; GGA induced Hsp70 but not Hsp90, Hsc70, Hsp60,
Hsp32 and protected tubular epithelial cell from apoptosis, inturn
ameliorated tubular damage by I/R injury in rat, induced Hsp70 and
CAS REGISTRY NUMBERS:
57-13-6 biological studies, Blood nitrogen; geranylgeranylacetone
decreased blood urea nitrogen following ischemia reperfusion injury in
rat
7722-84-1 biological studies, geranylgeranylacetone induced Hsp70,
protected tubular epithelial cell from apoptosis, inturn ameliorated
tubular damage by I/R injury in rat and induced Hsp70 rat tubular
epithelial cell culture
60-27-5 geranylgeranylacetone decreased serum creatinine levels after
ischemia reperfusion injury in rat
143-33-9 154-17-6 geranylgeranylacetone induced Hsp70, protected tubular
epithelial cell from apoptosis, inturn ameliorated tubular damage by
I/R injury in rat and induced Hsp70 rat tubular epithelial cell culture
6809-52-5 117-39-5 GGA induced Hsp70, decreased BUN, serum creatinine and
protected tubular epithelial cell from apoptosis, inturn ameliorated
tubular damage by I/R injury in rat, induced Hsp70 and suppressed
apoptosis in rat tubular epithelial cell culture

17/7/80 (Item 4 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

143057357 CA: 143(4)57357x JOURNAL
Mouse models incorporating alterations in the major tumor suppressor genes P53 and P16: their use in screening for potential carcinogens, developing further relevant mouse models, and screening for potential chemopreventive and chemotherapetutic agents
AUTHOR(S): Lubet, Ronald; Wang, Yian; Zhang, Zhongqiu; You, Ming
LOCATION: Chemoprevention Agent Development Research Group, National Cancer Institute, Rockville, MD, USA
JOURNAL: Exp. Lung Res. (Experimental Lung Research) DATE: 2005
VOLUME: 31 NUMBER: 1 PAGES: 117-133 CODEN: EXLRDA ISSN: 0190-2148

LANGUAGE: English PUBLISHER: Taylor & Francis, Inc.
SECTION:
CA214000 Mammalian Pathological Biochemistry
CA203XXX Biochemical Genetics
CA204XXX Toxicology
IDENTIFIERS: review tumor suppressor gene screening carcinogen chemopreventive chemotherapetutic agent
DESCRIPTORS:
Gene,animal...
CDKN2A; potential use of mutant mice in screening for carcinogens as well as preventive or therapeutic agents and pros, cons with germline mutation in tumor suppressor genes P16/Ink4AARFlocus in devel Disease models... Mus musculus... Mutation... Carcinogens... p53(protein)
...
potential use of mutant mice in screening for carcinogens as well as preventive or therapeutic agents showed relevance of dominant-neg. P53 and pros and cons with germline mutation in tumor suppressor Cyclin dependent kinase inhibitors...
p16INK4A; potential use of mutant mice in screening for carcinogens as well as preventive or therapeutic agents and pros, cons with germline mutation in tumor suppressor genes P16/Ink4AARFlocus in dev Gene,animal...
TP53; potential use of mutant mice in screening for carcinogens as well as preventive or therapeutic agents showed relevance of dominant-neg. P53 and pros and cons with germline mutation in tumor supp Gene,animal...
tumor suppressor; potential use of mutant mice in screening for carcinogens as well as preventive or therapeutic agents showed relevance of dominant-neg. P53 and pros and cons with germline mutation i

17/7/81 (Item 5 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

134142618 CA: 134(11)142618s JOURNAL
Structure and mutation analysis of the gene encoding DNA fragmentation factor 40 (caspase-activated nuclease), a candidate neuroblastoma tumor suppressor gene

AUTHOR(S): Judson, Hannah; Van Roy, Nadine; Strain, Lisa; Vandesompele,
Jo; Van Gele, Mireille; Speleman, Frank; Bonthron, David T.
LOCATION: Molecular Medicine Unit, St. James's University Hospital,
University of Leeds, Leeds, UK, LS9 7TF
JOURNAL: Hum. Genet. DATE: 2000 VOLUME: 106 NUMBER: 4 PAGES:
406-413
CODEN: HUGEDQ ISSN: 0340-6717 LANGUAGE: English PUBLISHER:
Springer-Verlag
SECTION:
CA203003 Biochemical Genetics
CA213XXX Mammalian Biochemistry
CA214XXX Mammalian Pathological Biochemistry
IDENTIFIERS: gene DFFB structure DNA fragmentation factor 40 human,
caspase activated nuclease gene DFFB structure human,
neuroblastoma
tumor suppressor gene mutation human DFFB
DESCRIPTORS:
Gene,animal...
DFFB; structure and mutation anal. of human gene DFFB encoding DNA
fragmentation factor 40 (caspase-activated nuclease) suggest that
DFFB
is not a neuroblastoma tumor suppressor gene
Genetic element...
exon, organization of DFFB gene; structure and mutation anal. of
human
gene DFFB encoding DNA fragmentation factor 40 (caspase-activated
nuclease) suggest that DFFB is not a neuroblastoma tumor suppre
Chromosome...
human 1, localization of DFFB gene; structure and mutation anal.
of
human gene DFFB encoding DNA fragmentation factor 40
(caspase-activated
nuclease) suggest that DFFB is not a neuroblastoma tumor sup
Chromosome...
human 9, localization of a DFFB pseudogene; structure and mutation
anal. of human gene DFFB encoding DNA fragmentation factor 40
(caspase-activated nuclease) suggest that DFFB is not a
neuroblastoma t
Genetic polymorphism... Mutation...
in DFFB gene; structure and mutation anal. of human gene DFFB
encoding
DNA fragmentation factor 40 (caspase-activated nuclease) suggest
that
DFFB is not a neuroblastoma tumor suppressor gene
Genetic element...
intron, organization of DFFB gene; structure and mutation anal. of
human gene DFFB encoding DNA fragmentation factor 40
(caspase-activated
nuclease) suggest that DFFB is not a neuroblastoma tumor supp
Nerve,neoplasm...
neuroblastoma, tumor suppressor gene; structure and mutation
anal. of

human gene DFFB encoding DNA fragmentation factor 40 (caspase-activated
nuclease) suggest that DFFB is not a neuroblastoma tumor s
Genetic mapping...
of DFFB gene and a DFFB pseudogene; structure and mutation anal. of
human gene DFFB encoding DNA fragmentation factor 40 (caspase-activated
nuclease) suggest that DFFB is not a neuroblastoma tumor sup
Gene,animal...
pseudogene, DFFB-like; structure and mutation anal. of human gene DFFB
encoding DNA fragmentation factor 40 (caspase-activated nuclease)
suggest that DFFB is not a neuroblastoma tumor suppressor gene
Genomic imprinting...
structure and mutation anal. of human gene DFFB encoding DNA
fragmentation factor 40 (caspase-activated nuclease) suggest that DFFB
is not a neuroblastoma tumor suppressor gene
Gene,animal...
tumor suppressor, neuroblastoma; structure and mutation anal. of human
gene DFFB encoding DNA fragmentation factor 40 (caspase-activated
nuclease) suggest that DFFB is not a neuroblastoma tumor suppre
CAS REGISTRY NUMBERS:
208939-71-3 gene DFFB; structure and mutation anal. of human gene DFFB
encoding DNA fragmentation factor 40 (caspase-activated nuclease)
suggest that DFFB is not a neuroblastoma tumor suppressor gene

17/7/82 (Item 6 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

131318574 CA: 131(24)318574d PATENT
Cloning vectors not using antibiotic resistance markers for use in lactic
acid bacteria in food processing
INVENTOR(AUTHOR): Sorensen, Kim Ib; Larsen, Rasmus; Johansen, Eric
LOCATION: Den.
ASSIGNEE: Chr. Hansen A/S
PATENT: PCT International ; WO 9954488 A1 DATE: 19991028
APPLICATION: WO 99DK209 (19990414) *DK 98551 (19980421) *US 82555 (19980421)
PAGES: 61 pp. CODEN: PIXXD2 LANGUAGE: English
PATENT CLASSIFICATIONS:
CLASS: C12N-015/74A; C12N-015/68B; C12N-009/88B; C12N-009/48B
DESIGNATED COUNTRIES: AE; AL; AM; AT; AT; AU; AZ; BA; BB; BG; BR; BY; CA;
CH; CN; CU; CZ; CZ; DE; DE; DK; DK; EE; EE; ES; FI; FI; GB; GD; GE; GH; GM;

HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV;
MD; MG; MK; MN; MW; MX; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SK; SL;
TJ; TM; TR; TT; UA; UG; US; UZ; VN; YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU;
TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS; MW; SD; SL; SZ; UG; ZW; AT; BE
; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ;
CF; CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG
  SECTION:
    CA203002 Biochemical Genetics
    CA210XXX MICROBIAL, ALGAL, AND FUNGAL BIOCHEMISTRY
    CA217XXX Food and Feed Chemistry
  IDENTIFIERS: Lactococcus cloning vector suppressor mutation marker, supD
    marker cloning vector Lactococcus, lactic acid bacteria cloning vector
    suppressor mutation marker
  DESCRIPTORS:
Mutation...
    amber, suppressible, as selectable marker; cloning vectors not using
    antibiotic resistance markers for use in lactic acid bacteria in food
    processing
tRNA...
    amber suppressor, as selectable marker; cloning vectors not using
    antibiotic resistance markers for use in lactic acid bacteria in food
    processing
Lysins...
    bacteriolysins, gene for, expression in Lactococcus of; cloning vectors
    not using antibiotic resistance markers for use in lactic acid bacteria
    in food processing
Food...
    batter, genetic manipulation of lactic acid bacteria for processing of;
    cloning vectors not using antibiotic resistance markers for use in
    lactic acid bacteria in food processing
Bifidobacterium... Lactic acid bacteria... Lactobacillus... Lactococcus
lactis diacetylactis... Lactococcus lactis lactis... Lactococcus lactis...
Lactococcus... Leuconostoc... Pediococcus... Streptococcus...
    cloning vectors not using antibiotic resistance markers for use in
    lactic acid bacteria in food processing
Dough... Fruit and vegetable juices... Meat... Musts... Vegetable... Wine

bacteria in food processing
Gene,microbial...
supD, as selectable marker; cloning vectors not using antibiotic resistance markers for use in lactic acid bacteria in food processing
Gene,microbial...
supE gene, as selectable marker; cloning vectors not using antibiotic resistance markers for use in lactic acid bacteria in food processing
Gene,microbial...
supF, as selectable marker; cloning vectors not using antibiotic resistance markers for use in lactic acid bacteria in food processing
Gene,microbial...
supP, as selectable marker; cloning vectors not using antibiotic resistance markers for use in lactic acid bacteria in food processing
Mutation... tRNA...
suppressor, as selectable marker in lactic acid bacteria; cloning vectors not using antibiotic resistance markers for use in lactic acid bacteria in food processing
Gene,microbial...
supU, as selectable marker; cloning vectors not using antibiotic resistance markers for use in lactic acid bacteria in food processing
Gene,microbial...
supZ, as selectable marker; cloning vectors not using antibiotic resistance markers for use in lactic acid bacteria in food processing

CAS REGISTRY NUMBERS:

289-95-2 auxotrophy for, as selectable marker in lactic acid bacteria; cloning vectors not using antibiotic resistance markers for use in lactic acid bacteria in food processing
9001-92-7 9025-40-5 9031-41-8 9031-99-6 9068-81-9 9074-83-3
54249-88-6 gene for, expression in Lactococcus of; cloning vectors not using antibiotic resistance markers for use in lactic acid bacteria in food processing
1414-45-5 gene for synthesis of or resistance to, expression in Lactococcus of; cloning vectors not using antibiotic resistance markers for use in lactic acid bacteria in food processing
55467-39-5 pepN gene for, expression in Lactococcus of; cloning vectors not using antibiotic resistance markers for use in lactic acid bacteria in food processing
249270-40-4 249270-41-5 249270-42-6 249270-43-7 249270-44-8

249270-45-9 249270-46-0 249270-47-1 249270-48-2 249270-49-3
249270-50-6 249270-51-7 249270-52-8 249270-53-9 249270-54-0
249270-55-1 249270-56-2 249270-57-3 249270-58-4 249270-59-5
249270-60-8 249270-61-9 249270-63-1 249270-64-2 249270-65-3
unclaimed nucleotide sequence; cloning vectors not using antibiotic
resistance markers for use in lactic acid bacteria in food processing
249270-62-0 unclaimed protein sequence; cloning vectors not using
antibiotic resistance markers for use in lactic acid bacteria in food
processing
248583-15-5 unclaimed sequence; cloning vectors not using antibiotic
resistance markers for use in lactic acid bacteria in food processing

17/7/83 (Item 7 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

131014777 CA: 131(2)14777u JOURNAL
Regulation of cytochrome P-450 (CYP) 1B1 in mouse Hepa-1 variant cell
lines: a possible role for aryl hydrocarbon receptor nuclear translocator
(ARNT) as a suppressor of CYP 1B1 gene expression
AUTHOR(S): Eltom, Sakina E.; Zhang, Leying; Jefcoate, Colin R.
LOCATION: Center for Environmental Toxicology and Department of Pharmacology, University of Wisconsin Medical School, Madison, WI, USA
JOURNAL: Mol. Pharmacol. DATE: 1999 VOLUME: 55 NUMBER: 3 PAGES: 594-604 CODEN: MOPMA3 ISSN: 0026-895X LANGUAGE: English PUBLISHER: American Society for Pharmacology and Experimental Therapeutics
SECTION:
CA203004 Biochemical Genetics
CA206XXX General Biochemistry
CA213XXX Mammalian Biochemistry
IDENTIFIERS: cytochrome P4501B1 regulation mouse Hepa1 variant cell line,
aryl hydrocarbon receptor nuclear translocator suppressor CYP1B1 gene
expression
DESCRIPTORS:
Promoter(genetic element)...
ARNT shows inhibitory effect on proximal; regulation of cytochrome P
450 (CYP) 1B1 in mouse Hepa-1 variant cell lines indicates possible
role for aryl hydrocarbon receptor nuclear translocator (ARNT)
Enhancer(genetic element)...
ARNT shows stimulatory effects in; regulation of cytochrome P 450 (CYP)

1B1 in mouse Hepa-1 variant cell lines indicates possible role for aryl
hydrocarbon receptor nuclear translocator (ARNT) as supp
Proteins,specific or class...
aryl hydrocarbon receptor nuclear translocator; regulation of
cytochrome P 450 (CYP1B1) in mouse Hepa-1 variant cell lines indicates
role for ARNT as suppressor of CYP 1B1 expression
Gene,animal...
CYP1A1, mRNA levels in LA2 were extremely low and unresponsive to TCDD;
regulation of cytochrome P 450 (CYP1B1) in mouse Hepa-1 variant cell
lines indicates role for ARNT as suppressor of CYP 1B1 expr
mRNA...
CYP1B1 mRNA and protein were expressed at levels seen in TCDD-induced
WT; regulation of cytochrome P 450 (CYP1B1) in mouse Hepa-1 variant
cell lines indicates role for ARNT as suppressor of CYP 1B1 ex
Gene,animal...
CYP1B1; regulation of cytochrome P 450 (CYP) 1B1 in mouse Hepa-1
variant cell lines indicates possible role for aryl hydrocarbon
receptor nuclear translocator (ARNT) as suppressor of CYP 1B1 gene expr
Animal cell line...
Hepa 1, ARNT-defective variant, CYP1B1 and CYP1A1 different expression
in; regulation of cytochrome P 450 (CYP1B1) in mouse Hepa-1 variant
cell lines indicates role for ARNT as suppressor of CYP 1B1 e
Mouse...
regulation of cytochrome P 450 (CYP) 1B1 in mouse Hepa-1 variant cell
lines indicates possible role for aryl hydrocarbon receptor nuclear
translocator (ARNT) as suppressor of CYP 1B1 gene expression
Transcriptional regulation...
repression; regulation of cytochrome P 450 (CYP) 1B1 in mouse Hepa-1
variant cell lines indicates possible role for aryl hydrocarbon
receptor nuclear translocator (ARNT) as suppressor of CYP 1B1 gene
Enhancer(genetic element)...
xenobiotic-responsive element (XRE) 1/2 and XRE4, formed
TCDD-unresponsive complexes; regulation of cytochrome P 450 (CYP1B1) in
mouse Hepa-1 variant cell lines indicates role for ARNT as suppressor o
CAS REGISTRY NUMBERS:
9035-51-2 biological studies, 1B1; regulation of cytochrome P 450 (CYP)
1B1 in mouse Hepa-1 variant cell lines indicates possible role for aryl

hydrocarbon receptor nuclear translocator (ARNT) as suppressor of CYP
1B1 gene expression
1746-01-6 CYP1B1 mRNA and protein were expressed at levels seen in TCDD-induced WT; regulation of cytochrome P 450 (CYP1B1) in mouse Hepa-1 variant cell lines indicates role for ARNT as suppressor of CYP
1B1 expression

17/7/84 (Item 8 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

130247725 CA: 130(19)247725v JOURNAL
Identification of the mouse neuromuscular degeneration gene and mapping
of a second site suppressor allele
AUTHOR(S): Cox, Gregory A.; Mahaffey, Connie L.; Frankel, Wayne N.
LOCATION: The Jackson Laboratory, Bar Harbor, ME, 04609, USA
JOURNAL: Neuron DATE: 1998 VOLUME: 21 NUMBER: 6 PAGES: 1327-1337
CODEN: NERNET ISSN: 0896-6273 LANGUAGE: English PUBLISHER: Cell Press
SECTION:
CA203003 Biochemical Genetics
CA214XXX Mammalian Pathological Biochemistry
IDENTIFIERS: mouse neruomuscular degeneration gene identification mapping
suppressor allele
DESCRIPTORS:
DNA helicases...
ATPase-, nmd mutation as transcriptional activator and ATPase/DNA helicase; identification of the mouse neuromuscular degeneration gene
and mapping of a second site suppressor allele
Mouse... Muscle atrophy...
identification of the mouse neuromuscular degeneration gene and mapping
of a second site suppressor allele
Mutation...
nmd, transcriptional activator and ATPase/DNA helicase previously described as Smbp2, Rip1, Gf1, or Catf1; identification of the mouse
neuromuscular degeneration gene and mapping of a second site supp
Genes(animal)...
Nmd; identification of the mouse neuromuscular degeneration gene and
mapping of a second site suppressor allele
Genetic mapping... Mouse chromosome 13... Phenotypes...
severity of nmd phenotype is attenuated in a semidominant fashion by
locus on chromosome 13; identification of the mouse neuromuscular

degeneration gene and mapping of a second site suppressor allele
Deletion(mutation)...
single amino acid, in nmdJ, and splice donor mutation in nmd2J;
identification of the mouse neuromuscular degeneration gene and mapping
of a second site suppressor allele
Genes(animal)...
Smbp2, Rip1, Gf1, or Catf1, mutated gene identified as nmd previously
described as; identification of the mouse neuromuscular degeneration
gene and mapping of a second site suppressor allele
Motor neurons...
spinal, degeneration of; identification of the mouse neuromuscular
degeneration gene and mapping of a second site suppressor allele
CAS REGISTRY NUMBERS:
9000-83-3 -DNA helicase, nmd mutation as transcriptional activator and
ATPase/DNA helicase; identification of the mouse neuromuscular
degeneration gene and mapping of a second site suppressor allele

17/7/85 (Item 9 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

127315579 CA: 127(23)315579r PATENT
Designing genes that can be used to replace a number of deleterious alleles and their use with extragenic suppressors in gene therapy
INVENTOR(AUTHOR): Farrar, Gwenyth Jane; Humphries, Peter; Kenna, Paul
Francis
LOCATION: Ire.,
ASSIGNEE: Provost, Fellows and Scholars of the College of the Holy and
Undivided Trini; Farrar, Gwenyth Jane; Humphries, Peter; Kenna, Paul
Francis
PATENT: PCT International ; WO 9737014 A1 DATE: 19971009
APPLICATION: WO 97GB929 (19970402) *GB 966961 (19960402)
PAGES: 89 pp. CODEN: PIXXD2 LANGUAGE: English
PATENT CLASSIFICATIONS:
CLASS: C12N-015/11A; C12N-009/00B; A61K-048/00B; A61K-031/70B; C07H-021/00B
DESIGNATED COUNTRIES: AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; CA; CH; CN;
CU; CZ; DE; DK; EE; ES; FI; GB; GE; GH; HU; IL; IS; JP; KE; KG; KP; KR; KZ;
LC; LK; LR; LS; LT; LU; LV; MD; MG; MK; MN; MW; MX; NO; NZ; PL; PT; RO; RU;
SD; SE; SG; SI; SK; TJ; TM; TR; TT; UA; UG; US; UZ; VN; YU; AM; AZ; BY; KG;
KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; KE; LS; MW; SD; SZ; UG; AT; BE

; CH; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF;
CG; CI; CM; GA; GN; ML; MR; NE; SN; TD; TG
  SECTION:
    CA203002 Biochemical Genetics
  IDENTIFIERS: gene therapy allele suppression replacement, synthetic gene
    replacement design wobble base, ribozyme suppression mutant allele
    expression
  DESCRIPTORS:
Synthetic genes...
    animal, for replacement of mutant allele; designing genes that can be
    used to replace no. of deleterious alleles and their use with
    extragenic suppressors in gene therapy
Gene therapy...
    designing genes that can be used to replace no. of deleterious alleles
    and their use with extragenic suppressors in gene therapy
Antibodies... Antisense DNA... Peptide nucleic acids... Ribozymes...
    for suppression of mutant allele expression; designing genes that can
    be used to replace no. of deleterious alleles and their use with
    extragenic suppressors in gene therapy
Peptides,biological studies...
    inhibitory, for suppression of mutant allele expression; designing
    genes that can be used to replace no. of deleterious alleles and their
    use with extragenic suppressors in gene therapy
Rhodopsins...
    mutant allele of gene for, specific ribozyme cleavage of; designing
    genes that can be used to replace no. of deleterious alleles and their
    use with extragenic suppressors in gene therapy
Proteins(specific proteins and subclasses)...
    peripherins (eye rod outer segment), mutant allele of gene for,
    specific ribozyme cleavage of; designing genes that can be used to
    replace no. of deleterious alleles and their use with extragenic supp
Genes(animal)...
    synthetic, for replacement of mutant allele; designing genes that can
    be used to replace no. of deleterious alleles and their use with
    extragenic suppressors in gene therapy
Oligonucleotides...
    triple helix-forming, for suppression of mutant allele expression;
    designing genes that can be used to replace no. of deleterious alleles
    and their use with extragenic suppressors in gene therapy
Collagens,biological studies...
    type 1A2, mutant allele of gene for, specific ribozyme cleavage of;

designing genes that can be used to replace no. of deleterious alleles
and their use with extragenic suppressors in gene therapy

17/7/86 (Item 10 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

127273843 CA: 127(20)273843q JOURNAL
IS911-mediated intramolecular transposition is naturally temperature sensitive
AUTHOR(S): Haren, Laurence; Betermier, Mireille; Polard, Patrice; Chandler, Michael
LOCATION: Laboratoire de Microbiologie et Genetique Moleculaires, CNRS UPR9007, 31062, Toulouse, Fr.
JOURNAL: Mol. Microbiol. DATE: 1997 VOLUME: 25 NUMBER: 3 PAGES: 531-540 CODEN: MOMIEE ISSN: 0950-382X LANGUAGE: English PUBLISHER: Blackwell
SECTION:
CA203005 Biochemical Genetics
CA207XXX Enzymes
CA210XXX MICROBIAL, ALGAL, AND FUNGAL BIOCHEMISTRY
IDENTIFIERS: IS911 mediated intramol transposition temp sensitive
DESCRIPTORS:
Insertion sequence...
IS911; IS911-mediated intramol. transposition is naturally temp. sensitive
Circular DNA...
IS911-mediated intramol. transposition at 30°C was sufficiently high to permit detection in vivo of an excised circular form of defective single IS911 chromosomal copy when OrfAB is supplied in
Transposition(genetic)...
IS911-mediated intramol. transposition is naturally temp. sensitive
Transposases...
OrfAB; IS911-mediated intramol. transposition at 30°C was sufficiently high to permit detection in vivo of an excised circular
form of defective single IS911 chromosomal copy when OrfAB is supp
Heat effects(biological)...
transposition was greatly reduced at 42°C compared with 37°C; IS911-mediated intramol. transposition is naturally temp. sensitive
Point mutation...
two point mutants of OrfAB rendered reactions partially temp. resistant; IS911-mediated intramol. transposition is naturally temp.
sensitive

17/7/87 (Item 11 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

122153276 CA: 122(13)153276g JOURNAL
Transcription factor ATF2 regulation by the JNK signal transduction pathway
AUTHOR(S): Gupta, Shashi; Campbell, Debra; Derijard, Benoit; Davis, Roger J.
LOCATION: Med. Sch., Univ. Massachusetts, Worcester, MA, 01605, USA
JOURNAL: Science (Washington, D. C.) DATE: 1995 VOLUME: 267 NUMBER: 5196 PAGES: 389-93 CODEN: SCIEAS ISSN: 0036-8075 LANGUAGE: English
SECTION:
CA203004 Biochemical Genetics
CA213XXX Mammalian Biochemistry
IDENTIFIERS: Jun protein kinase pathway regulation ATF2, transactivator
ATF2 regulation JNK signal transduction
DESCRIPTORS:
Phosphorylation,biological...
ATF2 was phosphorylated by c-Jun amino-terminal protein kinase on two closely spaced threonine residues within the NH2-terminal activation domain
Mutation,substitution...
replacement of threonine residue phosphorylation sites with alanine within the NH2-terminal activation domain of c-Jun amino-terminal protein kinase inhibited the transcriptional activity of ATF2
Gene,animal... Phosphoproteins,gene E1A...
threonine to alanine mutations within the NH2-terminal activation domain of c-Jun amino-terminal protein kinase also inhibited ATF2-stimulated gene expression mediated by the retinoblastoma tumor supp
Ribonucleic acid formation factors,ATF-2 (activating transcription factor 2)... Signal transduction,biological...
transcription factor ATF2 regulation by the c-Jun amino-terminal protein kinase signal transduction pathway
Eye,neoplasm, retinoblastoma...
tumor suppressor; mutations of phosphorylation sites within the NH2-terminal activation domain of c-Jun amino-terminal protein kinase inhibited ATF2-stimulated gene expression mediated by retinoblasto
CAS REGISTRY NUMBERS:
72-19-5 biological studies, ATF2 was phosphorylated by c-Jun amino-terminal protein kinase on two closely spaced threonine residues

```
    within the NH2-terminal activation domain
155215-87-5  transcription factor ATF2 regulation by the c-Jun
    amino-terminal protein kinase signal transduction pathway
?
PLEASE ENTER A COMMAND OR BE LOGGED OFF IN 5 MINUTES
? ds

Set     Items   Description
S1        581   (ATTENUAT? OR AVIRULENT OR VACCIN?) AND SALMONELLA
AND (PAI
              OR LEUX OR PATHOGENICITY(W)ISLAND)
S2        174   RD S1  (unique items)
S3        139   S2 NOT PY>2006
S4          0   S3 AND (LEUX OR TRNA5LEU)
S5          1   S3 AND TRNA
S6          0   S3 AND SUPP
S7        462   (ATTENUAT? OR AVIRULENT OR VACCIN?) AND (TYPHI OR
DUBLIN OR
              TYPHIMURIUM) AND (PAI OR LEUX OR PATHOGENICITY(W)ISLAND)
S8        163   RD S7  (unique items)
S9          1   S8 AND (LEUX OR TRNA OR SUPP)
S10       113   E1-E5
S11        46   AU='COHEN, P. S.'
S12       159   S10 OR S11
S13         3   S12 AND (LEUX OR TRNA OR SUPP)
S14      3901   (LEUX OR TRNA5LEU OR SUPP)
S15      3349   RD S14  (unique items)
S16      2662   S15 NOT PY>2005
S17        87   S16 AND (DELET? OR MUTAT? OR VARIANT? OR MUTEIN OR
AVIRULE-
             NT OR ATTENUAT?)
?

---Logging off of Dialog---


? logoff
       19jun08 11:16:13 User226352 Session D1072.3
            $7.68    1.241 DialUnits File5
              $78.08  32 Type(s) in Format  7
           $78.08  32 Types
    $85.76  Estimated cost File5
            $2.80    0.372 DialUnits File6
               $2.66  1 Type(s) in Format  2
            $2.66  1 Types
     $5.46  Estimated cost File6
            $4.26    0.660 DialUnits File24
              $27.00  10 Type(s) in Format  7
           $27.00  10 Types
    $31.26  Estimated cost File24
           $48.55    1.824 DialUnits File34
              $30.96  4 Type(s) in Format  7
```

```
       $30.96  4 Types
$79.51  Estimated cost File34
        $0.73    0.098 DialUnits File40
 $0.73  Estimated cost File40
        $0.83    0.128 DialUnits File41
 $0.83  Estimated cost File41
        $1.24    0.238 DialUnits File45
           $2.16  1 Type(s) in Format  7
        $2.16  1 Types
 $3.40  Estimated cost File45
        $2.14    0.449 DialUnits File50
 $2.14  Estimated cost File50
        $3.28    0.768 DialUnits File65
           $9.10  7 Type(s) in Format  7
        $9.10  7 Types
$12.38  Estimated cost File65
        $7.06    0.702 DialUnits File71
          $24.50  10 Type(s) in Format  7
       $24.50  10 Types
$31.56  Estimated cost File71
       $13.65    1.062 DialUnits File73
          $24.85  7 Type(s) in Format  7
       $24.85  7 Types
$38.50  Estimated cost File73
        $0.75    0.170 DialUnits File98
 $0.75  Estimated cost File98
        $1.68    0.259 DialUnits File103
 $1.68  Estimated cost File103
        $0.75    0.116 DialUnits File136
           $2.70  1 Type(s) in Format  7
        $2.70  1 Types
 $3.45  Estimated cost File136
        $0.47    0.152 DialUnits File143
 $0.47  Estimated cost File143
        $4.93    0.964 DialUnits File144
          $11.52  6 Type(s) in Format  7
       $11.52  6 Types
$16.45  Estimated cost File144
        $4.39    1.247 DialUnits File155
           $0.96  4 Type(s) in Format  7
        $0.96  4 Types
 $5.35  Estimated cost File155
        $2.23    0.363 DialUnits File156
           $4.32  4 Type(s) in Format  7
        $4.32  4 Types
 $6.55  Estimated cost File156
        $1.07    0.229 DialUnits File162
 $1.07  Estimated cost File162
        $1.45    0.113 DialUnits File172
 $1.45  Estimated cost File172
        $1.19    0.092 DialUnits File305
 $1.19  Estimated cost File305
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            $0.80    0.220 DialUnits File369
               $1.57  1 Type(s) in Format  7
            $1.57  1 Types
     $2.37  Estimated cost File369
            $0.47    0.128 DialUnits File370
               $1.62  1 Type(s) in Format  7
            $1.62  1 Types
     $2.09  Estimated cost File370
            $0.34    0.119 DialUnits File393
     $0.34  Estimated cost File393
           $16.72    1.279 DialUnits File399
              $41.72  14 Type(s) in Format  7
           $41.72  14 Types
    $58.44  Estimated cost File399
            $4.28    0.161 DialUnits File434
     $4.28  Estimated cost File434
            OneSearch, 26 files, 13.153 DialUnits FileOS
     $9.33  TELNET
   $406.79  Estimated cost this search
   $406.82  Estimated total session cost  13.533 DialUnits
Logoff: level 05.22.00 D  11:16:13
```